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THE RESULTS OF THE BOSTON CHEST X-RAY SURVEY*

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BOSTON

COMMUNITY-WIDE x-ray surveys had been conducted in eight cities of the United States of over 100,000 population, in co-operation with the Division of Tuberculosis of the United States Public Health Service, before a similar program was planned for the City of Boston. In all the locations where the program had been carried out, experience had proved that to conduct a successful mass x-ray program, it would be necessary to have a well planned blueprint of operations, complete community participation, a well publicized activity, a large number of trained personnel and substantial available local funds, all in addition to the facilities lent by the United States Public Health Service. An effort of this scope could be justified only if tuberculosis in Boston was a serious disease. That it was the fourth leading cause of death was borne out by statistics and served as the impetus of the program.

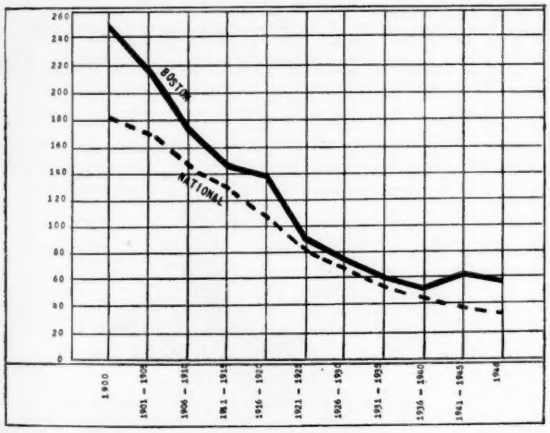

FIGURE 1. *National and Boston Death Rates from Pulmonary Tuberculosis, Five-Year Averages (1901-1945).*

Figure 1 demonstrates the five-year average for tuberculosis mortality as experienced nationally from 1900. Running more or less parallel to this national average is the five-year average for Boston. The national trend has declined, persistently, from approximately 180 per 100,000 population, in 1900, to below 40 per 100,000 population. Boston has had a higher rate than the national one, although there has been a steady decline from nearly 250 per 100,000 population at the beginning of the century to the present rate of approximately 60 per 100,000. This need not be too disturbing, since it is accepted that larger cities have a greater rate than rural communities.

Figure 2 shows that the rate for Boston is greater than the rate for most other large American cities. Statisticians may arrive at conclusions that are favorable owing to the manner in which the data are assembled in relation to allocation, variations in population in color and age distribution, or exclusion or inclusion of veterans, which may alter the final figures. Regardless of these factors, however, there has been too much tuberculosis in Boston.

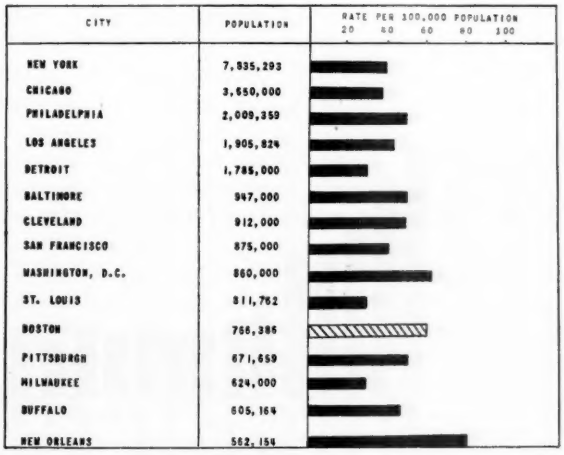

FIGURE 2. *Tuberculosis Death Rate for Larger Cities in the United States for 1947.*

Figure 3 shows that Boston has a greater rate than almost all the listed eastern cities in the United States.

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These unfavorable comparisons should not discourage one too greatly, for there are some very hopeful figures (Fig. 4). In 1900 Boston had a rate of 250 per 100,000 population. There has been a constant betterment until the advent of World War II, when the rate increased slightly. Now, however, the former trend has been resumed. It is almost too early to make any predictions for 1950. It is interesting to note, however, that for the first four months of 1949, there were 124 deaths in Boston due to tuberculosis, whereas, for the first four months of 1950, there have been but 98 deaths. In that period, but 271 cases of tuberculosis were reported in 1949, whereas 343 cases have been reported in 1950. There is no doubt that the mass chest x-ray survey has been instrumental in detecting cases of tuberculosis earlier and has thus led to a lessening mortality and a greater number of reported cases.

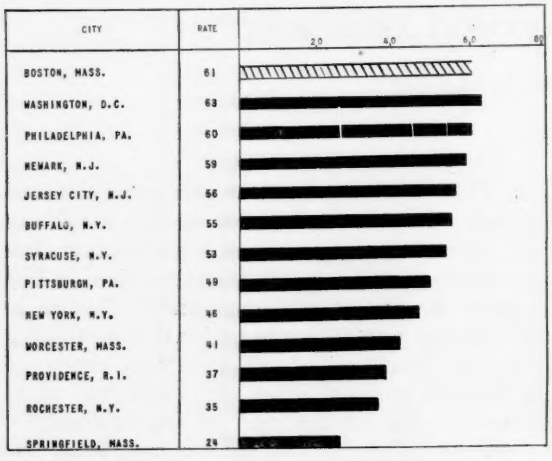

FIGURE 3. *Death Rates from Pulmonary Tuberculosis in Larger Eastern Cities, Five-Year Averages (1942-1946).*

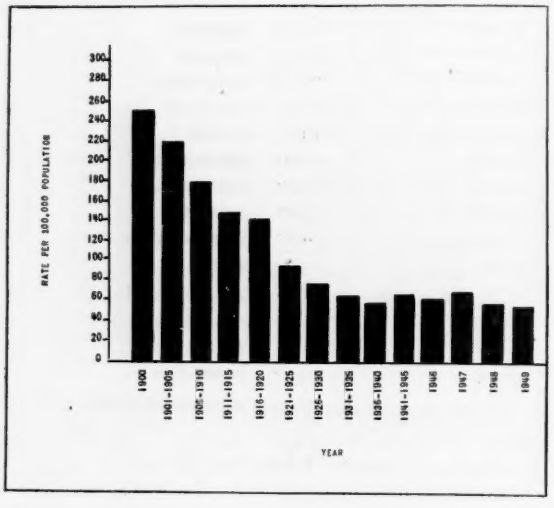

FIGURE 4. *Death Rates from Pulmonary Tuberculosis, 1900-1949 (Five-Year Averages, 1901-1945).*

Boston has a population of about 750,000. Table 1 shows the distribution of the population according to white, nonwhite and Chinese. About 3 per cent of the population are nonwhite, and about 1/5 of 1 per cent are Chinese. Both the case rate and the death rate of the Chinese and of the nonwhite are extremely high.

TABLE 1. *Case Rate and Death Rate for Pulmonary Tuberculosis According to Race, 1945-1949 (Five-Year Average).*

RACE	POPULATION	NEW CASE RATE per 100,000 population	DEATH RATE per 100,000 population
Chinese	1,671	1292.6	550.5
Nonwhite	23,679	300.6	169.7
White	741,036	102.5	52.8
Total	766,386		

The diagram map of Boston (Fig. 5) is of the greatest value in localizing the problem geographically. The density of the shading indicates the magnitude of the incidence of tuberculosis for the year 1949. Records show that there has been no great variation for many years. The South End, Roxbury, the West End and Charlestown have been the main problem centers. The greatest effort was made in these areas to see that every person was examined; no effort was spared in any section, but in these particular areas, the effort was intensified.

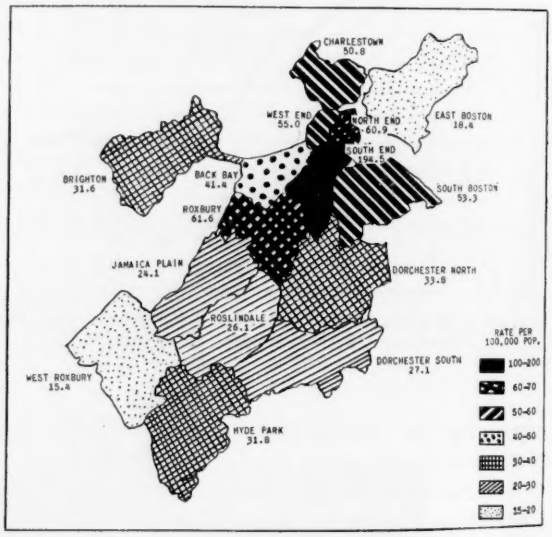

FIGURE 5. *Mortality from Pulmonary Tuberculosis in Boston in 1949 by Sections of the City.*

It was the goal of the planning group to examine over 500,000 persons during the program. Figure

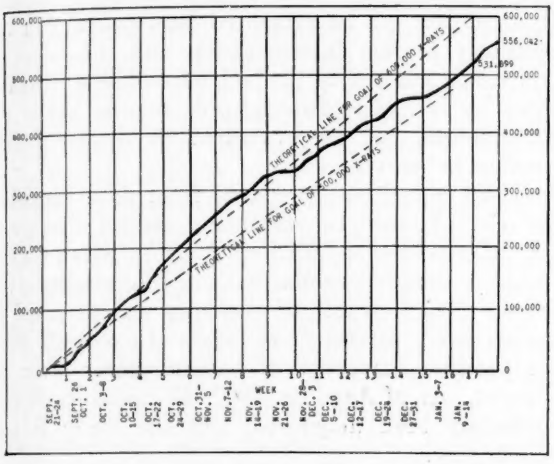

FIGURE 6. *Cumulative X-Ray Production by Weeks.*

6 indicates the progress that was made week by week. Except for the first two weeks at the start of the program, the number of x-ray films taken exceeded the goal set. It was possible to continue in operation one downtown unit two weeks beyond the originally planned sixteen-week period and during these two weeks over 4000 examinations were made at this single unit. In addition, while the survey was in progress, over 20,030 films were taken at several large hospitals in Boston where 70-mm. equipment was available. This brought the total to 556,042. At the completion of the program, 536,012 70-mm. x-ray films had been taken and interpreted by the roentgenologists, exclusive of the 20,030 taken in Boston hospitals.

Table 2 shows the interpretation placed upon the 70-mm. films. It will be noted that more than 97 of every 100 films taken were apparently negative. Of the smaller x-ray films 14,465 showed thoracic abnormalities. Of these, 8070 gave evidence of tuberculosis or suspected tuberculosis, and the remainder showed other intrathoracic abnormalities. During the entire period when the smaller films were being taken, a retake center was operated on Boston Common. All persons who showed evidence of tuberculosis, or other chest diseases, were requested to return to this center for a standard 14 by 17 x-ray film. At the same time, a résumé of their clinical history was taken.

Table 3 catalogues the result of the x-ray interpretations of all 9372 14-x-17 x-ray films that were taken. There were thoracic abnormalities in 7254; 4122 gave evidence of tuberculosis; 443 were considered suspicious of tuberculosis; and 2406

TABLE 2. *70-Mm.-Film Breakdown by X-Ray Impression.*

BREAKDOWN	NUMBER	FILMS TAKEN	FILMS INTERPRETED	THORACIC ABNORMALITIES
		%	%	%
Unsatisfactory	7,071	1.32	—	—
Films interpreted	528,941	98.68	100.00	—
Examinations apparently negative	514,476	—	97.27	—
Evidence of thoracic abnormality	14,465	—	2.73	100.00
Tuberculosis	4,003	—	0.76	27.67
Suspected tuberculosis	4,067	—	0.77	28.12
Nontuberculous chest disease	3,045	—	0.58	21.05
Cardiac conditions	3,313	—	0.57	22.90
Unclassified	37	—	0.06	0.26

TABLE 4. *Boston Chest X-Ray Program Tentative Cancer Statistics.**

STATISTICS		No.
Total films taken in survey		536,012
Total "cancer suspects"		402
Total diagnoses to date (physicians and clinics)		177
Diagnoses other than cancer (about 30 per cent of 402)		125
Cases of cancer (roughly 1 per 10,000 films)		52
Primary (1 per 20,000 films)	25	
Successful operation	11	
Inoperable (1 known, 1 suspected before Survey	14	
Presumptive diagnosis of primary cancer†	12	
Metastatic	12	
Other types of tumors (1 lymphoma, 1 lymphosarcoma, 1 case of Hodgkin's disease)	3	

*Information obtained from Dr. Clarence L. Scamman, medical director, Massachusetts Division, Inc., of American Cancer Society, as of April 18, 1950.

†Six refused further study; further study not advised in 3; 3 still under study and observation.

TABLE 3. *Film Breakdown by X-Ray Impression.*

BREAKDOWN	NUMBER	FILMS INTERPRETED	THORACIC ABNORMALITY	TUBERCULOSIS	70-MM. FILMS
		%	%	%	%
Films interpreted	9,372	100.00	—	—	—
Examinations apparently negative	2,118	22.60	—	—	—
Thoracic abnormality	7,254	77.40	100.00	—	1.37
Tuberculosis	4,122	—	56.82	100.00	0.78
Minimal	2,780	—	—	67.44	—
Moderately advanced	1,152	—	—	27.95	—
Far advanced	87	—	—	2.11	—
Stage unknown	103	—	—	2.50	—
Suspected tuberculosis	443	—	6.11	—	0.08
Nontuberculous chest disease	2,406	—	33.17	—	0.45
Cardiac condition	283	—	3.90	—	0.05

represented cases of nontuberculosis chest diseases. From these 14-x-17 films, a diagnosis of cardiac disease was made in 283 cases. No effort is made here to indicate the extent of cardiac conditions determined as a result of the taking of 70-mm. films or the mass chest x-ray program. Dr. David D. Rutstein, in a personal communication, has stated that a representative sample of 31,091 70-mm. photofluorographic films taken for tuberculosis in the Boston chest x-ray survey were examined independently by two groups of readers for the existence of an abnormal cardiac silhouette. Information will be presented later concerning the accuracy of film interpretation and the effect of color, age and sex on the incidence of heart disease. The 70-mm. photofluorographic film as taken for tuberculosis will be evaluated as a case-finding method in heart disease.

Table 4 had been prepared from data furnished by Dr. Clarence L. Scamman, medical director of the Massachusetts Division of the American Cancer Society. Briefly summarized, the information given shows that 1 cancer of the lung was detected in about every 10,000 films taken. In all, 402 films of patients suspected of having cancer were reviewed. As of April 18, 1950, 52 cancer cases had definitely been diagnosed, and the study is still under way.

In addition to tuberculosis, heart disease and cancer, innumerable abnormal chest conditions were discovered as a result of the taking of more than 500,000 70-mm. x-ray films and more than 9000 standard 14-x-17 films. The roentgenologists, who read the large and the small plates, listed several hundred abnormal and interesting conditions. These abnormalities have been catalogued, and patients

TABLE 5. *Data among 247 Newly Reported Active Mass-Survey Cases in Boston Residents, October 6, 1949, to May 1, 1950.**

Datum	No. of Cases	Percentage
Sex:		
Males	180	72.9
Females	67	27.1
Total	247	
Stage of disease:		
Minimal	69	27.9
Moderately advanced	102	41.3
Far advanced	50	20.3
Not stated	26	10.5
Total	247	
Source of report:		
Hospital	37	15.0
Private physician	48	19.4
Diagnostic clinic	129	52.2
Other (other health-department clinics, Veterans Administration)	33	13.4
Total	247	

*At the time of the original report, 95, or 38.5 per cent, had positive sputums.

TABLE 6. *Classification of 247 Newly Reported Active Cases in Boston Residents by Age Group and Sex.*

Age	Total Cases		Males		Females	
	no.	percentage	no.	percentage	no.	percentage
yr.						
Under 15	2	0.8	1	0.5	1	1.5
15–24	27	10.9	9	5.0	18	26.9
25–34	55	22.3	36	20.0	19	28.4
35–44	58	23.6	47	26.1	11	16.4
45–54	46	18.2	35	19.5	11	16.4
55–64	31	12.7	27	15.0	4	6.0
65–74	26	10.7	23	12.8	3	4.4
75 and over	2	0.8	2	1.1	0	—
Totals	247		180		67	

TABLE 7. *Findings by Age Group and District among Cases of Pulmonary Tuberculosis in Boston Chest X-Ray Program.*

District	Patients under 15		Patients 15–24		Patients 25–34		Patients 35–44		Patients 45–54	
	males	females	males	females	males	females	males	females	males	females
East Boston	—	—	1	1	3	2	3	—	—	—
Charlestown	—	—	—	—	1	—	3	—	2	—
North End	—	—	—	—	1	1	1	—	—	—
West End	1	—	—	—	1	—	2	1	1	—
Back Bay	—	—	1	—	2	—	2	—	3	1
South End	—	—	1	2	7	7	8	3	8	—
South Boston	—	—	1	3	3	2	3	—	1	—
Roxbury	—	—	1	4	6	3	9	—	10	—
Dorchester North	—	—	1	6	6	—	8	2	1	2
Dorchester South	—	—	1	1	—	2	2	—	2	3
West Roxbury	—	—	—	—	—	—	—	—	—	1
Brighton	—	—	1	1	—	2	4	2	3	1
Jamaica Plain	—	—	1	—	—	—	2	—	1	—
Roslindale	—	—	—	—	3	1	1	—	1	3
Hyde Park	—	1	—	—	2	—	—	—	1	—
Totals	1	1	9	18	35	20	48	8	34	11
	2		27		55		56		45	

requiring treatment have been referred to their private physicians or to clinics. Many of the conditions found have been extremely valuable for teaching purposes, and lantern slides of these films have been prepared. As a direct result of the mass x-ray program, 247 active cases of tuberculosis among Boston residents have been reported. In an analysis of the residence of persons examined, it was shown that slightly more than 60 per cent were Boston residents.

Table 5 shows the stage of the disease in the cases of tuberculosis reported as a result of the survey. It also gives the source of the report of the case. At the time of the original report, 38.5 per cent of the patients had a positive sputum.

Table 6 gives the age distribution of the 247 cases of tuberculosis and the distribution by sex.

Table 7 lists the geographical locations from which the active cases of tuberculosis came. In the South End section of the city, where tuberculosis was most prevalent, the greatest number of cases was found. Roxbury, a high-rate district, came second. The West End, in proportion to the population, had a relatively high case-finding rate. By and large, the results produced were as expected. Not only was the greater number of cases in proportion to the population found in the areas where tuberculosis was most prevalent, but also the age distribution was about as anticipated. The case-finding program is still continuing, and each day additional cases are being reported. What has happened to the cases diagnosed is of great importance. Table 8 shows the disposition of the patients. As the program was planned, concern was expressed regarding the disposition of the patients who would be found. It has been possible to hospitalize most patients through the co-operation of the Boston City Hospital, the Sanatorium Division of the Boston City Hospital, the Massachusetts Department of Public Health, the Veterans' Administration and several local sanatoriums. At the present time, because enough nurses cannot be found to staff tuberculosis institutions, there is a short waiting list for patients requiring hospitalization. Facilities at the Boston Sanatorium, in Mattapan, are being extended, as are those of several of the State institutions. To care for a limited number of patients at the main hospital, the Boston City Hospital is remodeling some of its facilities and is thus making it possible to segregate tuberculous patients.

TABLE 8. *Disposition of 247 Newly Reported Active Mass-Survey Cases in Boston Residents, October 6, 1949, to May 1, 1950.*

DISPOSITION*	NO. OF CASES	PERCENTAGE
Sanatorium	134	54.2
Discharged from sanatorium	10	4.1
With permission	6	—
Against advice	4	—
Clinic (including Veterans Administration)	70	28.3
Private physician	22	8.9
Moved from city	1	0.4
Deaths	10	4.1
Total	247	

*May 1, 1950.

The conduct of the mass chest x-ray survey required great effort. At present the statistical data obtained as a result of the survey are being catalogued in co-operation with the United States Public Health Service.

When the survey was begun it was planned to examine all persons over fifteen years of age who lived, worked, shopped or visited in Boston. More than 500,000 films were taken. Approximately 60 per cent of all persons examined were residents of Boston; except for a very small percentage who came from practically every state in the Union as well as from Canada, Mexico and other foreign countries, the remaining 40 per cent were residents of Massachusetts.

TABLE 7 (*Concluded*).

DISTRICT	PATIENTS 55–64		PATIENTS 65–74		PATIENTS 75 AND OVER		TOTALS	
	MALES	FEMALES	MALES	FEMALES	MALES	FEMALES	MALES	FEMALES
East Boston	—	—	—	—	—	—	7	3
Charlestown	—	—	1	—	—	—	7	0
North End	—	—	1	—	—	—	3	1
West End	6	—	3	1	—	—	14	2
Back Bay	2	—	1	—	—	—	11	1
South End	7	—	7	—	1	—	39	12
South Boston	3	1	1	—	—	—	12	6
Roxbury	3	2	5	1	1	—	33	10
Dorchester North	2	1	3	—	—	—	21	11
Dorchester South	1	—	2	1	—	—	8	7
West Roxbury	—	—	—	—	—	—	0	1
Brighton	2	—	—	—	—	—	10	6
Jamaica Plain	—	—	—	1	—	—	4	1
Roslindale	2	1	1	—	—	—	8	5
Hyde Park	—	—	—	—	—	—	3	1
Totals	28	5	23	4	2	0	180	67
	33		27		2		247	

In addition to the 247 active cases of tuberculosis reported in Boston as a result of the survey, cases of active tuberculosis have been reported from other cities and towns in Massachusetts. When the x-ray film of a nonresident was interpreted as showing tuberculosis, that fact was reported to the Massachusetts Department of Public Health, which in turn furnished the information to the district health officer and the local board of health. Data pertaining to the total number of tuberculosis cases reported to cities and towns are being computed. At the present time, it is not possible to determine, exactly, the cases reported as a result of the survey. It is expected that at the close of the present year, these data will be available. A large number of active cases of tuberculosis, a sizable number of cases of cancer, a substantial number of heart conditions and many other pathologic chest conditions were discovered as a result of the survey. It is particularly important to know that many of the cases of tuberculosis discovered were found in the early stage of the disease, when the possibility of obtaining successful treatment is more favorable.

OBLITERATION OF PAIN AT THE SITE OF REFERENCE BY INTRADERMAL INFILTRATION ANESTHESIA IN FIRST-STAGE LABOR*

Preliminary Report

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BOSTON

FOR many decades obstetricians have concerned themselves with finding newer drugs and combinations of drugs to alleviate the pain of the first stage of labor. The purpose of this paper is to suggest a method of relief of pain based on interference with the viscerosensory reflex. According to Reynolds,[1] "Our present knowledge of the uterine innervation consists of numerous facts, many of which appear to be contradictory." The focal point of the uterine innervation is the pelvic plexuses, which supply the viscera of the pelvic cavity and are situated at the sides of the rectum and the vagina. They are formed on either side by a continuation of the hypogastric plexus, by the sacrovisceral afferent and efferent fibers from the second and third and fourth sacral nerves and by a few filaments from the first two sacral ganglions. The nerves of the uterus leave the pelvic plexuses with strands that supply the bladder and the upper part of the vagina. The rectum, bladder, urethra and lower vagina receive separate innervation by way of the pudendal plexus.

There are no published data that reveal the course of the sensory nerves from the uterus to the central nervous system. The fact that resection of the hypogastric nerves alone relieves uterine pain shows them to pass through this part of the sympathetic system. It is noteworthy that sensory reflexes are abolished by section of the hypogastric nerves alone, although the sacral connections to the pelvic plexus are intact. Available evidence suggests that few motor and no sensory connections exist between the uterus and the sacral nerves.

Cleland[2] stated, "The pains of labor are made up of two components, namely, (a) that due to uterine contraction which is transmitted by afferent fibers through the eleventh and twelfth thoracic roots and (b) due to stretching of the birth canal which is transmitted through certain undetermined sacral roots." In 1928 Rose[3] described the successful intradermal use of novocain to break the pain-reference arc in the first stage of labor. Weiss and Davis,[4] in a series of experiments, concluded that visceral pain is in reality a viscerosensory reflex as stated by MacKenzie[5] and others. Painful sensations arise as a result of irritation of a viscus. Such irritation produces nervous impulses, which pass from the irritated viscera along the splanchnic nerves to certain segments within the spinal cord. The perpetual bombardment of this cord segment with visceral impulses gives rise to an "irritable focus in the cord." The normal afferent impulses arising from the skin then enter a region of the spinal cord that is abnormally irritable. These normal impulses under such conditions give rise to painful sensations, which are referred to the peripheral structures. Anatomically, cutaneous branches of the eleventh and twelfth thoracic nerves supply the skin of the lower hypogastric area of the abdomen near the midline. The lateral cutaneous branch arising from the twelfth nerve reaches the skin a short distance above the iliac crest, sending a filament medialward along the iliac crest, and then passes downward to the skin of the buttock. Both the iliohypogastric nerve and the ilioinguinal nerve communicate with the last thoracic nerve and supply the skin over the inguinal ligaments and the symphysis. The sacral nerves supply cutaneous branches over the sacrum and the sacroiliac joints.

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Close questioning of patients in first-stage labor reveals consistent reference of pain to the areas of the skin supplied by the nerves mentioned above.

Method

Abdominal pain was abolished upon infiltration of the skin with an anesthetic agent, starting at the midline just above the symphysis pubis and continuing the infiltration in a linear fashion intradermally, first to the left and then to the right above the inguinal ligaments, out to the anterior-superior

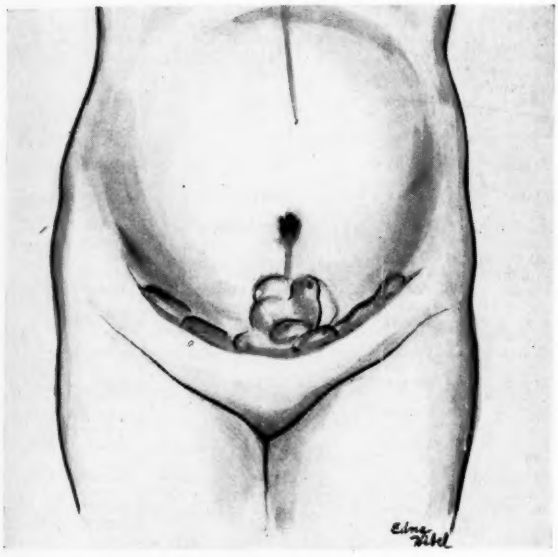

Figure 1. *Distribution of Wheals in the Lower Abdomen.*

iliac spines and then up the midline of the abdomen for a distance of 7.5 cm., extending the infiltration laterally on each side for a distance of 2.5 cm. (Fig. 1). When the patient complained of associated back pain, the skin area over the upper sacrum and over the sacroiliac joints was infiltrated intracutaneously (Fig. 2). Relief of pain in the back was not dramatic. In some it was abolished, but in most cases the pain was voluntarily described by the patients as an ache. If the backache were entirely referred, one would expect more uniform cessation of pain upon infiltration of the skin in this area, but this does not always occur. It is interesting that some patients complain of back pain early in labor even though the presenting part is unengaged. Pressure per se on the sacral plexus as a cause for back pain cannot be assumed. It is possible that some of this pain is due to stretching of the sacral plexus that must occur during the process of labor.

In the first group of cases 1 per cent novocain with epinephrine in 1:100,000 dilution was used effectively when injected as described above. Its activity lasted from two to three hours. Pontocaine, 0.1 per cent, did not produce any longer action. Most patients were treated with Metycaine, 1.5 per cent solution, to which epinephrine in 1:200,000 dilution was added. The action of this drug was found to be of longer duration, and in some cases as much as six hours of anesthesia was obtained. In most multiparas and in a good many of the primiparas, a single injection sufficed during the first stage of labor. Approximately 30 cc. of the solution was used for abdominal infiltration, and up to 30 cc. for the back infiltration. This drug can be reinjected without any untoward effects. The infiltration can be started as soon as the patient is in labor and complains of pains, regardless of the cervical dilatation of early labor, because it will not stop the progress of labor. In many of the patients of this series it was noted that when dilatation sufficient to admit three and a half to four

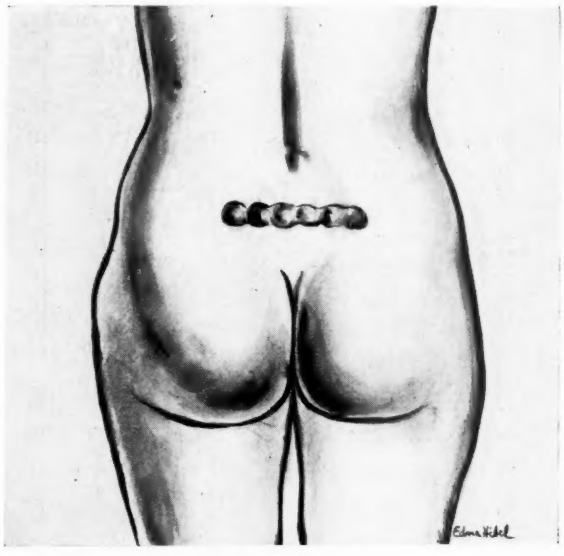

Figure 2. *Distribution of Wheals over the Sacrum and Sacroiliac Joints.*

fingers had been reached, patients complained of pain deep in the pelvis, indicating perhaps that at this stage of cervical dilatation the pain was not chiefly of uterine origin. In multiparas at this stage, second-stage anesthesia — as a single caudal injection, saddle block, pudendal block or nitrous oxide, oxygen and ether — could be given without interference with the course of labor. In primiparas scopolamine, 0.32 mg. intravenously, was given when unhindered progress for a longer time was desired.

Any idiosyncrasy to this drug can be ruled out within a short period by the observation of a small intracutaneous wheal. The question has been raised

whether or not absorption of the anesthetic agent used might produce an analgesic effect of a systemic nature. Inasmuch as these patients are wide awake during this method of treatment, one can readily determine sensitivity to the needle prick, which is revealed as sharp, except at the actual sites of infiltration. With infiltration of one side of the abdomen, these patients refer their pain to the opposite side.

The cases in this series were unselected. The ward patients were not informed what was being done or what it was hoped would be accomplished by the injection. Private patients were informed in detail about the procedure. Only the private patients who expressed a desire for this type of anesthesia, however, were subjected to the procedure. In all the cases reported, the anesthetic effectiveness of the drug was simultaneous with its administration, without causing any alteration of the tone of the uterus or the frequency, intensity or duration of the uterine contraction. Because of the cessation of pain with the patient conscious, all the patients became fully co-operative in the labor process. In only 1 case was the infiltration terminated because the patient became extremely nervous and anxious. This was a patient who was unable to take novocain in the dentist's chair and required general anesthesia for all her dental work. Many of the ward patients who were in active labor when the drug was administered, complaining severely of pain, became completely calm and expressed a desire to get out of bed and to walk around or to sit in a chair. All patients remained conscious of their contractions even though the painful sensations were absent. It is interesting that without any suggestion upon my part, these patients no longer spoke in terms of labor pains but referred to them correctly as contractions.

Of these 40 patients, 20 were ward patients who did not know what was being done or what the

TABLE 1. *Relief of Pain in 40 Cases.*

CASE NO.	AGE	PARITY	GRAVIDITY	TIME OF INFILTRATION	PRESENTING PART	STATION OF PRESENTING PART	DILATATION OF CERVIX
	yr.						
1	22	0	I	2 p.m.	Vertex	Middle	2 fingers
2	21	0	I	5 p.m.	Vertex	Middle	1½ fingers
3	28	II	III	3:45 p.m.	Full breech	Middle	2 fingers
4	27	I	II	6:30 p.m.	Vertex	Low middle	3 fingers
5	31	II	III	2 p.m.	Vertex	High middle	2 fingers
6	17	0	I	1 p.m.	Vertex	Middle	2 fingers
7	18	0	I	10 a.m., repeated at 2 p.m.	Vertex	Middle	2 fingers
8	23	0	I	3 p.m.	Vertex	Middle	2 fingers
9	19	0	I	4 p.m.	Vertex	Low middle	2 fingers
10	23	0	I	9:45 a.m.	Vertex	High	1 finger
11	32	I	II	10:30 p.m.	Vertex	Low middle	1½ fingers
12	16	0	I	3:20 p.m.	Vertex	Low middle	2½ fingers
13	26	I	II	2 a.m.	Vertex	Low middle	3 fingers
14	32	0	I	9:30 p.m.	Vertex	Low	2½ fingers
15	30	0	II	9 p.m.	Vertex	Middle	2 fingers
16	30	VI	VII	11:30 a.m.	Vertex	Middle	2 fingers
17	46	III	IV	4:45 p.m.	Vertex	High middle	1½ fingers
18	28	0	I	10 a.m.	Vertex	Middle	2½ fingers
19	25	0	II	11:30 a.m.	Vertex	Middle	1½ fingers
20	32	II	III	12:30 p.m.	Vertex	Middle	1 finger
21	28	II	III	4 a.m.	Vertex	Middle	1½ fingers
22	27	0	I	9:15 a.m.	Vertex	Middle	1½ fingers
23	29	I	IV	9:30 a.m.	Vertex	Low middle	1 finger
24	28	I	II	11:30 p.m.	Vertex	Low	1½ fingers
25	26	I	II	3:30 a.m.	Vertex	Middle	1 finger
26	27	0	I	9 a.m.	Vertex	High middle	1 finger
27	28	I	II	4 a.m.	Vertex	Middle	1 finger
28	30	I	II	3:30 p.m.	Vertex	Middle	1 finger
29	28	I	II	7:30 a.m.	Vertex	Middle	2 fingers
30	29	I	II	10 a.m.	Vertex	Middle	1½ fingers
31	38	0	I	11 a.m.	Vertex	Middle	1 finger
32	27	I	II	11:30 p.m.	Vertex	Low	2 fingers
33	31	V	VI	11 a.m.	Vertex	Middle	2 fingers
34	27	III	IV	11:15 a.m.	Vertex	Low	3 fingers
35	23	I	II	4:05 p.m.	Vertex	Middle	1½ fingers
36	17	0	I	10:30 a.m.	Vertex	Low middle	2½ fingers
37	35	III	IV	11 a.m.	Vertex	Middle	2½ fingers
38	31	0	I	2 p.m.	Vertex	Middle	3 fingers
39	30	I	II	9:45 a.m.	Vertex	Middle	2 fingers
40	29	0	I	11 a.m.	Vertex	High	1 finger

purpose of the procedure was. The remaining 20 were private cases.

The only criterion used for administration of the medication was the presence of painful contractions, regardless of the state of dilatation of the cervix. Twenty-two patients, of all degrees of parity, had complete relief of pain, as indicated by proximity to the time of actual delivery in these cases, second-stage anesthesia, such as that listed in Table 1, was given with no interference in the course of labor or in the dispatch with which the second stage of labor was completed. In the primiparas the pelvic pressure complained of occurred most frequently at dilatation admitting four fingers,

TABLE 1 (*Concluded*).

CASE NO.	DURATION OF ANESTHESIA	ADDITIONAL ANALGESIA DURING LABOR	RELIEF OF PAIN	SECOND-STAGE ANESTHESIA	TIME OF DELIVERY	TYPE OF DELIVERY
1 ...	5 hr.	None	++++	Spinal	7 p.m.	Low forceps
2 ...	2 hr., 45 min.	None	++++	Spinal	8 p.m.	Low forceps
3 ...	1 hr.	None	++++	Open ether	5 p.m.	Double footling extraction
4 ...	2 hr., 15 min.	None	++++	None	8:45 p.m.	Low forceps
5 ...	3 hr.	100 mg. of Demerol	+++	Ether	7 p.m.	Low forceps
6 ...	3 hr., 45 min.	None	++++	Spinal	4:45 p.m.	Low forceps
7 ...	12 hr.	Demerol and Seconal after 12 hr.	++++ for 12 hr.	Spinal 32 hr. after infiltration	—	Mid forceps
8 ...	4 hr.	None	++++	Spinal	7 p.m.	Low forceps
9 ...	3 hr.	None	++++	Spinal	7 p.m.	Low forceps
10 ...	4 hr., 15 min.	Demerol and Seconal at 3 fingers' dilatation	++	Spinal	9 p.m.	Low forceps
11 ...	1 hr., 15 min.	None	+++	Nitrous oxide, oxygen and ether	12 m.	Low forceps
12 ...	2 hr.	20 cc. of Metycaine into caudal canal at 5:15 p.m.	+++	—	7:20 p.m.	Low forceps
13 ...	1 hr., 15 min.	Same at 3:30 a.m.	+++	—	4:15 a.m.	Scanzoni operation
14 ...	1 hr., 15 min.	None	++++	Pudendal block	10:55 a.m.	Low forceps
15 ...	2 hr., 30 min.	None	++++	Pudendal block	11:50 p.m.	Low forceps
16 ...	1 hr., 30 min.	None	++++	Precipitate delivery	1 p.m.	Normal
17 ...	2 hr., 15 min.	100 mg. of Demerol	+++	Nitrous oxide, oxygen and ether	8:06 p.m.	Low forceps
18 ...	3 hr.	Pudendal block at 4 fingers' dilatation	+++	—	1:50 p.m.	Normal
19 ...	2 hr., 10 min.	None	++++	Single caudal injection at 1:40 p.m. (20 cc.)	2 p.m.	Low forceps
20 ...	4 hr.	None	++++	Pudendal block; nitrous oxide at 4:30 p.m.	5:05 p.m.	Normal
21 ...	4 hr.	None	++++	Saddle block	8 a.m.	Low forceps
22 ...	3 hr.	None	++++	Saddle block	12:30 p.m.	Mid forceps
23 ...	2 hr., 45 min.	None	++++	Nitrous oxide, oxygen and ether	2:30 p.m.	Low forceps
24 ...	3 hr.	None	+++	Spinal	2:50 p.m.	Scanzoni operation
25 ...	2 hr., 15 min.	None	+++	Saddle block	6 a.m.	Low forceps
26 ...	4 hr., 30 min.	None	+++	Saddle block	2 p.m.	Mid forceps
27 ...	3 hr.	None	+++	Saddle block	7:30 a.m.	Low forceps
28 ...	6 hr.	None	+++	Saddle block	10 p.m.	Low forceps
29 ...	1 hr., 30 min.	None	+++	Saddle block	9:15 a.m.	Low forceps
30 ...	3 hr.	Nitrous oxide, oxygen and ether with pains	+++	Nitrous oxide, oxygen and ether	4:30 p.m.	Low forceps
31 ...	5 hr.	Scopolamine, 0.3 mg. intravenously repeated once	++	Nitrous oxide, oxygen and ether	7 p.m.	Mid forceps
32 ...	2 hr., 30 min.	None	++++	Saddle block	2:30 a.m.	Normal
33 ...	2 hr.	None	++++	Nitrous oxide, oxygen and ether	1:30 p.m.	Normal
34 ...	3 hr.	100 mg. of Demerol at 4 fingers' dilatation	+++	Spinal	4 p.m.	Normal
35 ...	1 hr., 30 min.	100 mg. of Demerol at 3½ fingers' dilatation	+	Spinal	6 p.m.	Low forceps
36 ...	4 hr.	None	++++	Spinal	3 p.m.	Normal
37 ...	2 hr.	None	++++	Nitrous oxide, oxygen and ether	2 p.m.	Normal
38 ...	5 hr.	None	++++	Spinal	7 p.m.	Low forceps
39 ...	1 hr.	None	++++	Pudendal block and perineal infiltration	11:15 a.m.	Normal
40 ...	5 hr.	Scopolamine, 0.3 mg. intravenously at 4 p.m.	+++	Spinal	5 p.m.	Low forceps

"++++" in the column headed "Relief of Pain" in Table 1. Fifteen patients had "+++" relief (the presence of backache sufficient to cause the patient to mention it). Two patients had fair relief of pain "++," indicating painful backache requiring the use of some medication as the labor progressed. One patient had little relief of the abdominal or back pain, as indicated by "+."

It is interesting that almost all the multiparas complained of deep pelvic pressure with each contraction with dilatation sufficient to admit three and a half or four fingers, radiating in some cases down the anterior surface of the thigh. Because of at which time, if the progress of labor was active and the presenting part rotated, second-stage anesthesia was administered. In the cases in which rotation was not complete, the patient was given either 100 mg. of Demerol intramuscularly or scopolamine, 0.32 mg., intravenously. These medications were repeated on several occasions when the progress of labor was slow, and second-stage anesthesia was withheld so as not to inhibit progress. It appears from the findings discussed above that a new painful stimulus that is not of uterine origin occurs when the cervix is dilated sufficiently to admit three and a half or four fingers in most par-

turient patients. It also seems that the pain of first-stage labor up to that point is chiefly of uterine origin, following the pathways of Cleland; this pain was abolished completely in 55 per cent of the cases in this series and almost completely in an additional 37 per cent. Only in 3 cases were the results negligible.

It might be fair to assume that stretching of the surrounding tissues is the cause of pain in late first-stage and second-stage labor with the added discomfort from direct pressure upon the nerve roots of the sacral plexus.

In this series 23 low-forceps deliveries were performed. There were 4 midforceps deliveries, 2 Scanzoni maneuvers for persistent posterior presentation of heads, 2 breech deliveries and 9 normal deliveries. In Cases 11 and 17, labor was induced by intravenous administration of Pituitrin drip followed by intracutaneous infiltration anesthesia fifteen minutes after the start of the infusion, with relief of pain as noted. The patient in Case 10, who was described as having ++ relief of pain, was extremely apprehensive, with a psychotic background, and two weeks after delivery, a post-partum psychosis developed. I do not believe, in retrospect, that this type of case would be amenable to treatment of this sort, but with the background of anxiety, the patient presented a challenge for this treatment. Although apprehensive, she was most co-operative.

In Case 31 the patient, who was also apprehensive, remained in second-stage labor for almost three hours and failed to bring the vertex below the spines. Before dilatation sufficient to admit four fingers had been reached, she complained considerably of back pains. Case 35, in which relief was not obtained to any degree, is not explainable except that the labor was extremely active with rapid dilatation of the cervix.

Various types of second-stage anesthesia were used. In patients who did not want to be awake during the actual process of delivery, nitrous oxide, oxygen and ether was administered. Pudendal block, when performed during the latter part of first-stage labor, seemed to afford considerable relief of deep pelvic pain complained of at that stage. When block was performed together with perineal infiltration by means of Metycaine, painless delivery resulted.

Discussion

This clinical study seems to bear out the referred character of pain in first-stage labor, indicating a simple method whereby it can be relieved either completely or to a large extent. The effectiveness of the anesthesia is simultaneous with its administration, which in no way causes an alteration of the uterine tone or the frequency, intensity and duration of uterine contractions. It is too early to draw any conclusion regarding the time element in these cases. However, there is a strong suggestion that the actual process of labor for many patients was shorter than that in my usual experience. Whether the absence of systemic effects and the commonly encountered restlessness under sedation are related to this, I do not know. One thing that must be stressed as occurring uniformly in this group is the spontaneity with which the infants cried and the pinkness of their color immediately upon birth. This fact alone seems to warrant serious consideration for the welfare of the babies when large doses of sedation are given during labor.

It seems that parturient patients with cardiovascular-renal disease, impaired liver function and blood dyscrasias could best be relieved of pain in labor by such a method free of systemic effects. Certainly, in cases in which prematurity or sensitized babies of Rh– mothers are concerned, such a type of anesthesia, free of toxicity and depression upon the babies, becomes highly desirable. For the patients who wish the psychologic and intellectual satisfaction of childbirth and are unable to undertake the process naturally, this type of anesthesia is desirable.

Conclusions

Intradermal anesthesia is useful in first-stage labor in that pain during the greater part of the labor is abolished without the use of sedation with its depressing effects, particularly upon the baby. This type of anesthesia produces no local or systemic complications and requires no special knowledge or special aftercare. The technic of infiltration is simple and can be used in the home as well as in the hospital.

I am indebted to Drs. Frederick L. Good, Benjamin Tenney and Philip F. Snyder for their co-operation and help.

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FERROUS SULFATE TOXICITY*

Report of a Fatal Case

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FERROUS sulfate is generally considered a wholly innocuous drug. It is universally prescribed and with some abandon. The bulk of the research related to it has concerned its beneficial effects in anemias. Reports of toxicity are rare and, indeed, only 1 such case is to be found in the American literature of the last sixty years. Reports of 2 fatal cases in England stimulated descriptions of similar cases, with pertinent discussion and research. It seems important, therefore, to describe the following case in considerable detail — not only to draw attention to a drug that can be harmful and even lethal but also to promote understanding of the mechanisms of toxicity and its relation to the metabolism of iron, with the hope that wider discussion and research may be stimulated in the United States.

REVIEW OF THE LITERATURE

From 1850 to 1890 several cases of toxicity from iron compounds were reported.[1-6] References were made to severe gastrointestinal irritation and general collapse, but there was only 1 autopsied case. This had a typical gross picture.

In 1931 Hurst[7] described a case of iron encephalopathy following overdose of iron and ammonium citrate. Smith and Cook,[8] in 1934, mentioned a girl who swallowed 1.7 gm. (28 gr.) of ferrous sulfate and recovered.

In 1947 Forbes[9] reported 2 fatal cases of ferrous sulfate poisoning. In 1 of these a healthy boy, aged three years and three months, ate 50 0.2-gm. (3-gr.) tablets of an iron preparation containing also 2.5 mg. (1/25 gr.) of copper sulfate and 5.0 mg. (1/12 gr.) of manganese sulfate. He had two episodes of vomiting but seemed well for forty-eight hours, when he became jaundiced and restless, dying in fifty-three hours. Autopsy showed necrosis of the gastric and intestinal mucosas, slight renal tubular degeneration and a few pulmonary hemorrhages. In the other case a one-year-old boy swallowed 30 to 35 of the same pills. He had a hematemesis in an hour and was pale and showed signs of shock, with moist, labored breathing. In spite of immediate, vigorous treatment, he died in thirty hours. Autopsy showed bronchopneumonia, gastric and intestinal necrosis and degenerative changes in the liver, kidneys and pancreas. By animal experimentation, Forbes acquitted the copper and manganese traces as the toxic agents and established the cat lethal dose of ferrous sulfate as 0.065 gm. (1 gr.) per 64 gm. of body weight. Somers[10, 11] concurred after similar experiments.

Russell[12] mentioned that a number of pregnant women on massive iron therapy complained of headache, nausea and malaise and eventually manifested a mild albuminuria.

Thomson,[13] stimulated by these articles, reported 2 and later 6 cases of his own, all in children. They showed variously early pallor, drowsiness, vomiting, abdominal pain in 1 case, hematemesis in 4, positive guaiac or similar tests in 4 and sudden collapse and death in 2. Autopsy in 1 case demonstrated patchy atelectasis and severe gastric necrosis. The second showed much less severe necrosis.

Foucar, Gordon and Kaye[14] described a twenty-six-year-old man who ingested ¼ pound of ferrous sulfate (*USP*). He entered the hospital in shock and showed cyanosis, hematemesis and colic. In spite of gastric lavage, whole-blood transfusion and oxygen, he died in three hours. Autopsy revealed pulmonary hemorrhage and edema, erosion from the esophagus to the jejunum and congestion of the remaining bowel. Because the Prussian blue reaction was positive only in the tissues in direct proximity to the ferrous sulfate, the authors concluded that the iron "was important only as the vehicle of an anion that constituted a strong acid."

CASE REPORT

A 17-month-old girl was admitted to the hospital acutely cyanotic and unresponsive after ingestion of many ferrous sulfate tablets. The past history showed that she had been born after a normal delivery and that her health had been excellent until the evening of admission.

At 6 p.m. she was discovered to have ingested a handful of 0.3 gm. (5-gr.) ferrous sulfate tablets from a container she had found in the pocket of a coat hanging on a doorknob. She was apparently well until 10:00 p.m., when vomiting, followed by diarrhea, developed. At 1:00 a.m., because she was severely cyanotic, with vomiting and diarrhea, the family called a physician, who rushed the patient immediately to the hospital. In the vomitus 3 tablets, which had most of the enteric coating dissolved away and an estimated ⅛ of the ferrous sulfate itself absorbed, were found. In the stool 2 more partially digested tablets were obtained. These tablets had been prescribed for the mother about 4 days previously, and it was estimated that no more than 20 could be missing. At 2:00 a.m. the patient arrived at the hospital showing a gray cyanosis. She was completely limp and unresponsive, and her skin showed many blotchy ecchymoses. Coarse rhonchi, assumed to be due to aspirated vomitus, were audible over the right portion of the chest.

The blood pressure was unobtainable, and the respirations were slow and labored.

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A chest x-ray film showed slightly increased perihilar markings.

The child received 0.25 cc. of Coramine intravenously on admission, and a transfusion of 250 cc. of plasma was started. Although the respirations were adequately strong and there was no major obstruction of the airway, the cyanosis did not respond to oxygen. At 3:35 a.m. in the belief that she had a methemoglobinemia, 2.8 cc. of a 1 per cent solution of aqueous methylene blue was given, with a definite improvement in color and responsiveness. The blood type was AB, of which none was immediately available. A large specimen for serum iron determination was lost. At 4:30 a.m., while she was receiving 300 cc. of saline solution after the plasma, cyanosis was not entirely gone, and 1.2 cc. of methylene blue was given again in the tubing. At this time she was vomiting and raising fairly large amounts of bloody fluid through her nose. The black, watery stools, continuous since admission, were voluminous and contained oxyuris worms. No response was seen to the second injection of methylene blue, her respirations became less frequent (12 to 14 per minute) and stertorous, although there was little to aspirate, and at 5:10 a.m. she had a slight convulsion and died.

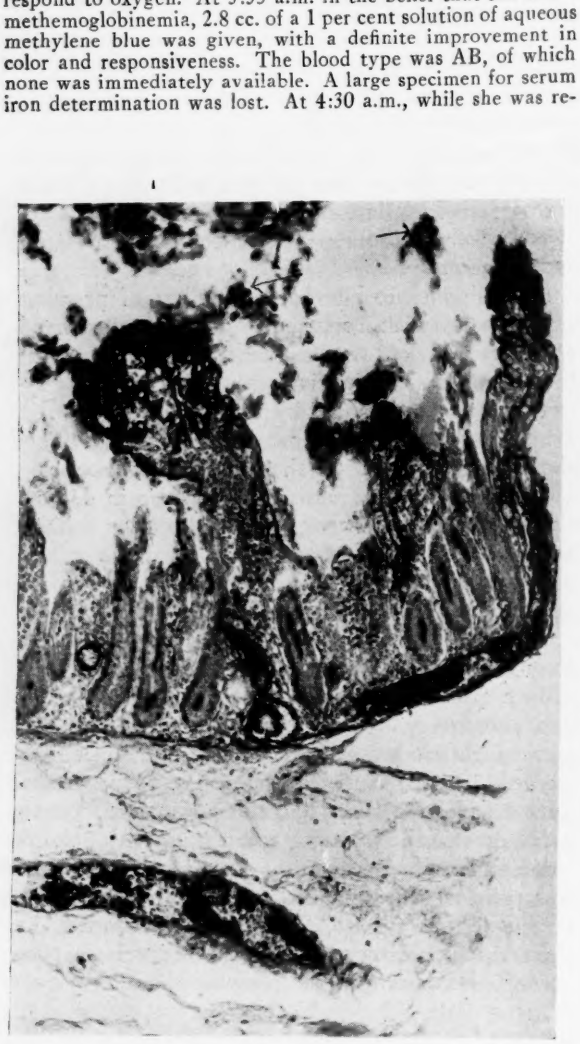

FIGURE 1. *Low-Power View of the Wall of the Ileum, Showing the Iron Pigment Stained Specifically by the Method of Turnbull (Tirmann–Schmelzer Reaction) (Black in the Photograph).*

The iron lies within strips of desquamated surface epithelium (see arrows), overlying and obscuring crests of the villi, and within capillaries and mucosal and submucosal veins. The iron pigment may also be seen in the lumens of the intestinal glands.

At post-mortem examination, performed 5 hours after death, the buccal mucosa and tongue were heavily coated with shaggy-appearing, dirty-brownish-gray material. The peritoneal cavity contained 200 cc. of clear, light-yellow fluid; the linings were smooth, thin and shiny. The pleural cavities were dry. The pericardium contained the usual amount of clear, straw-colored fluid.

The left lung weighed 68 gm. (usual weight, 64 gm.) and the right 98 gm. (usual weight, 66 to 72 gm.) They were crepitant in all lobes, but edematous. Small hemorrhages, averaging 0.8 cm. in diameter, were seen beneath the pleura and were wedge shaped, with the base directed toward the pleura. Though present in the upper and anterior areas, they were most numerous and prominent in the lower lobes and on the posterior surfaces. The mucosa of the larynx, trachea and bronchi to the smallest recognizable branches was covered by a somewhat mucoid, dirty-appearing, brownish-gray, thick, semisolid material.

The liver weighed 380 gm. (usual weight, 331 to 345 gm.). The cut surface had a mosaic appearance of pale, round areas surrounded by a narrow, bright-red zone.

The esophagus, stomach and small and large intestines contained a dirty-brownish-gray, metallic-appearing semisolid material, which was most prominent in the small bowel. The mucosa of the small intestine appeared necrotic, and large areas had sloughed away. When the metallic material was wiped gently away, pin-head-sized, round, regular areas of bright-red discoloration were seen in great numbers.

The unfixed brain weighed 1030 gm. (normal weight, 1010 to 1042 gm.). The surface vessels were very prominent. The subarachnoid fluid was definitely increased although there was no flattening of the convolutions. Several well demarcated, circular, reddish-brown areas 0.3 to less than 0.1 cm. in diameter were demonstrated in the caudate nuclei when the formalin-fixed brain was sectioned.

The heart weighed 50 gm. (normal weight, 48 to 52 gm.).

The spleen weighed 51 gm. (normal weight, 26 to 28 gm.). The follicles were prominent and the cut surface dark red.

The left kidney weighed 34.5 gm. (normal weight, 39 to 43 gm.) and the right 31.0 gm. (normal weight, 39 to 40 gm.). Both had slightly hyperemic surface vessels. The bladder contained no urine.

The adrenal glands were not remarkable.

Microscopical examination of the jejunal and ileal sections showed the surface epithelial layer to be completely eroded away. An occasional desquamated strip was seen, and its cells contained a brown granular pigment. The villous vessels, which were dilated and packed with red cells, contained a similar pigment, granular and usually golden brown though sometimes grayish brown. This pigment was distributed at the vessel periphery, apparently overlying or lying within the endothelial vessels. There was nowhere definite evidence of pigment within the actual cell walls. Irregular, clumped masses of the pigment were also found lying within the lumens of the vessels. The deeper and larger vessels of the mucosa and those of the submucosa were very prominent. The veins were numerous and engorged, containing larger pigment masses, fine granules at the center and coarser granules at the periphery of the masses. Again, some pigment was layered close to the wall and in the same questionable relation to the endothelium as in the villous vessels. There was no pigment in any arterial vessel.

In the lumens of the intestinal glands the pigment was also seen, but never within the cells lining them (Fig. 1 and 2). The lining cells were intact and viable in contradistinction to the sloughed, dying surface epithelial cells.

The mucosa of the ileum, and to a much lesser extent of the jejunum, was lightly and diffusely infiltrated with mononuclear cells, mainly of plasma-cell variety.

One section of ileum showed almost complete sloughing of the mucosa down to the muscularis mucosa, but this was not the usual picture. In another area there was extensive recent hemorrhage into the mucosa, with sloughing of the villi and intact mucosa beneath.

The lymphoid follicles revealed marked reticuloendothelial hyperplasia.

Sections of stomach demonstrated a loss of surface epithelium and a general appearance similar to that described in the small intestine, but to a much less extent. It is significant that the secreting cells of the gastric mucosa showed no pigment ingestion or damage.

The Turnbull blue method of staining for ferrous iron was used.[15] (It stains all reduced iron a deep blue.) This method

demonstrated that all pigment described in the routine hematoxylin and eosin preparations was iron (Fig. 1).

Two phenomena were revealed in the liver sections. Degeneration of the parenchymal cells involved the central four fifths of the lobule, evidenced by poorly staining, light pink, irregularly distributed granular cytoplasm and usually normal nuclei. (There were no fatty changes, and the bile ducts were not remarkable.) The second phenomenon was the presence of irregular masses of granular iron pigment in the lumens of the dilated portal veins (Fig. 3).

The Kupffer cells were large and viable and apparently did not contain iron. No granular iron masses were noted in the central veins.

Sections of kidney revealed small masses of granular material in the capsular space, and larger masses within the lumen of the tubules. The vessels contained none. This pigment stained a light blue with Turnbull's method, possibly because it was Zenker-formalin-fixed tissue, which is not as satisfactory for this method as tissue fixed in 10 per cent formalin.

Immediately surrounding the small terminal bronchioles in the lungs was an area of atelectasis. Peripheral to this

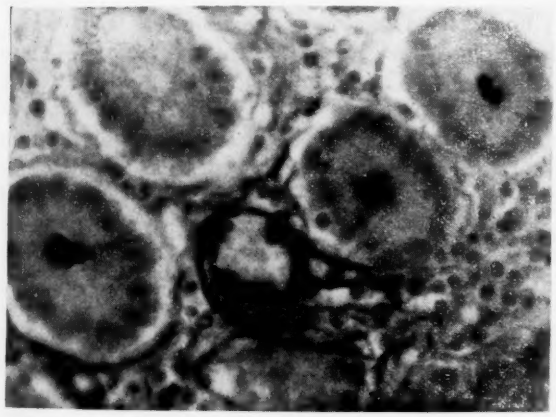

FIGURE 2. *A High-Power View of the Mucosa of the Ileum from the Same Section as That in Figure 1.*

The stained iron pigment is seen within the lumens of the glands. Note its absence within the normal-appearing epithelial cells lining them, as compared with the surface epithelial cells. There is a small vascular channel just off the center of the field, containing several irregular masses of iron pigment of varying sizes. This tendency to line up along the endothelium is characteristic of the iron in most capillaries and vessels studied.

was a zone of edema, congestion and hemorrhage, with obliteration of the normal alveolar architecture. There were only a few inflammatory cells. Some of the bronchioles contained red cells and fluid and were obstructed, infolded and ruptured.

Sections of heart disclosed prominent, increased, dilated and congested intramural capillaries.

Many representative sections of the brain were cut, including several from the caudate nucleus. There were no evident parenchymal changes, and no iron pigment could be demonstrated in the brain. Blood vessels, however, showed very prominent Virchow–Robin spaces, some containing a small amount of light pink, staining, very slightly granular material. All vessels were extremely engorged with blood.

Sections of the bone marrow, spleen and adrenal glands were not remarkable.

Analysis of the intestinal contents and the kidneys gave no evidence of heavy metals. No chemical analysis was done for iron.

Samples of blood taken before death for serum iron and methemoglobin determination were unfortunately lost. An analysis of blood taken after death for methemoglobin was done by Dr. Robertson, of the Biochemistry Department of the University of Vermont College of Medicine, using the method of Evelyn and Malloy.[16] The extinction of the band at 635 millimicrons is measured before and after quenching

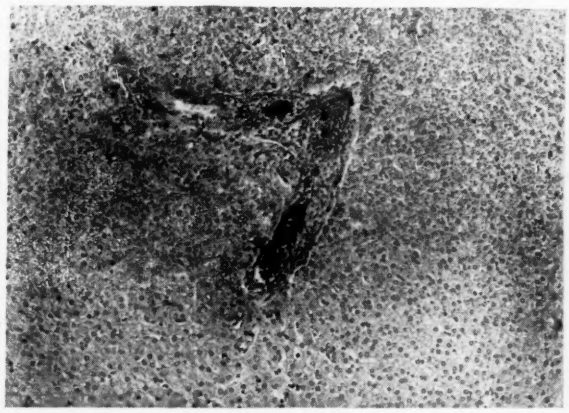

FIGURE 3. *Low-Power View of the Portal Area of the Liver, Showing Masses of Stained Iron Pigment (Black) Lying Within Slightly Dilated Portal Venous Channels.*

Iron could not be demonstrated in the central veins (not shown in the photograph) or in the sinusoids.

with cyanide. In this case the accuracy of the determination was impaired by the presence of methylene blue.

Some consideration was given to the capsule of the ferrous sulfate tablets. The manufacturers of the tablets furnished the information that they are a standard *U.S.P.* preparation and have a gastric-resistant coating. This is a universally used coating and consists of cane sugar with small quantities of starch, calcium, acacia, gelatin and shellac. The color is a certified food dye. All the coating constituents are considered to be therapeutically inert.

Discussion

Certain microscopical features are prominent. In the first place no iron pigment is seen in any vessels (or tissue) beyond the portal veins in the direction of blood flow except in the glomerular spaces and renal tubules. Specifically, none is seen in the central veins of the liver, nor in any of the Kupffer cells lining the sinusoids. The assumption is that the iron at this point goes into solution, presumably combined with serum protein.

In the second place, the assumption that there is a selective affinity of the intestinal mucosal cells for iron is strong. This is evidenced by the taking up of iron by the surface epithelial cells, which have died and have been sloughed off. On the other hand, iron pigment is seen consistently in the lumens of the intestinal glands, but none has been taken up by the glandular epithelium, which is viable and healthy. This selective capacity on the part of the intestinal mucosa is of considerable interest in the

light of the work of Hahn et al.[17] and Granick.[18, 19] The latter has suggested that a protein, apoferritin, is the hypothetical mucosal acceptor of iron of Hahn, producing ferritin. When the mucosal cells had been saturated with ferritin iron, the absorption of iron would be stopped until the body demand decreased the stores, and then absorption of iron would be resumed. Thus is postulated a mechanism of mucosal block or control of ferrous iron. We believe that the evidence presented above indicates the surface epithelial cells and not the glandular cells as the mucosal site of apoferritin and ferritin.

However, regarding the toxicity of ferrous iron, the "mucosal block" is not necessarily and, indeed, probably not effective when large amounts of iron are involved, as in the case reported above. It is suggested that the "barrier" of surface epithelium, with its controlling mechanism, is abolished by the initial nonspecific escharotic effect of sulfate and sulfurous compounds, thus allowing uncontrolled and very rapid absorption of the iron salt.

Gastric irritation is usually considered a characteristic of all oral iron preparations. Ferrous sulfate is no exception. The case reported above, as well as those in the literature, showed severe hemorrhagic gastritis and upper enteritis. Vomiting appeared early and became red, with gross blood. Autopsy showed generalized necrosis of the gastric mucosa. The black, watery, metallic-smelling diarrhea probably indicated irritation farther down as well. Foucar et al.[14] were unable to demonstrate gastrointestinal absorption of iron and therefore concluded that iron per se had no part in the mechanism of toxicity. The Prussian-blue method that they used demonstrated only ferric iron. Had they added Turnbull blue we believe they would have demonstrated the absorbed ferric form.

From the vomiting, diarrhea and enteric hemorrhage there is marked fluid loss. This may explain the shock-like state that the patients present. However, it has been suggested to us that large doses of iron, parenterally at least, can produce a nitritoid reaction. Heffter[20] refers to marked blood-vessel dilatation and capillary paralysis "as with arsenic." Mazur and Shorr[21] postulated a vasodepressor substance in certain types of shock and demonstrated that it was ferritin.

Clinically, the gray cyanosis, unresponsive to oxygen and very responsive to methylene blue, seems best explained as due to methemoglobinemia. Here, again, Heffter's work is corroborative. It states that there can be methemoglobin and hematin formation with both ferric and ferrous salts. We hope that the next observer will not lose his antemortem blood sample. The dual role[22] of methylene blue, reverting methemoglobin in small concentrations and producing it if the dose is greater, may explain the failure of the second dose in this case.

Questions about the effect of iron intoxication on the blood-clotting mechanism are raised by the scattered hemorrhages usually found at autopsy and the fact, in the case reported above, that the heart's blood was unclotted five hours after death.

Russell[12] pointed out evidence of renal impairment, and cloudy swelling of renal cells was described in this case.

Beyond general supportive measures and transfusion, the problem of proper therapy in ferrous sulfate toxicity is a difficult one.

BAL was not used because Randall and Seeler,[23] although not mentioning the ferrous ion, stated that the toxicity of certain metals is enhanced by combination with BAL. Work by Edge and Somers[24] on mice indicated that the iron combination has this effect, and its use by Thomson[25] was not beneficial. Somers[26] believes that Thomson's use of sodium bicarbonate lavage would be useful only immediately after ingestion and prefers a demulcent by mouth, aluminum hydroxide combined with bismuth sulfate and a central emetic.

The death described seems definitely to have been due to ferrous sulfate poisoning, and not to any capsular or other foreign ingredients. The strict directive that the pill bottle be kept out of reach of children should go with the dispensing of this, as well as with most pharmaceuticals. Perhaps the popular magazines should make the warning more widespread that even ferrous sulfate can be lethal.

The best management of these cases needs clarification. Intubation, gastric lavage and gastric precipitation of remaining drug is difficult in the face of severe vomiting, and perhaps is dangerous with the hemorrhage and necrosis present in the stomach. BAL is apparently not acceptable.

Whole blood is definitely indicated in the correction of this type of shock, and perhaps epinephrine would be the best stimulant.

If methemoglobinemia proves to be a regular part of the picture, Finch[27] recommends methylene blue as the perfect antidote, a fraction of a milligram to 10 mg. per kilogram of body weight. His advice was followed in the treatment of the case reported above. Whole-blood transfusions would, of course, help.

First aid in the home would probably be prompt emesis and feeding of raw eggs or milk, so that protein could absorb the iron. Hospitalization should be prompt, and there apomorphine would be available for early cases.

Summary

A fatal case of ferrous sulfate toxicity in a seventeen-month-old child, with autopsy findings, is presented. The patient exhibited signs of severe enteric irritation, vasomotor collapse, gray cyanosis unresponsive to oxygen but rapidly responsive to methylene-blue therapy, scattered hemorrhages and possible renal and pancreatic damage.

Iron absorption was shown by Turnbull-blue technic.

A hope is expressed that this report will stimulate the report of similar cases, and it is suggested that the toxicity of this drug, generally considered innocuous, be better publicized.

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THE ANTI-PITRESSIN FACTOR IN THE TREATMENT OF DYSMENORRHEA*

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DYSMENORRHEA, like other clinical syndromes whose etiology is obscure, has been considered by some a clinical state more appropriate for discussion by the psychiatrist than the gynecologist. The Ebers and Kahun papyri, thought to have been written 2000 B.C., refer to the dysmenorrheic syndrome. The Talmud and Ayur Veda, which antedate the Christian era, describe in detail the symptoms and treatment of dysmenorrhea. In every disease there is a psychic overlay that may exaggerate the subjective response of the patient, but this does not deny the underlying pathology or physiologic dysfunction involved. Primary dysmenorrhea is associated with a physiologic dysfunction of the myometrium that can be consistently demonstrated. There are frequently associated constitutional factors whose significance is just beginning to be understood.

Although this clinical syndrome is seen in all constitutional types the obese girl is usually spared the rigors of painful menstruation. The woman with genital hypoplasia as manifested by hypoplastic mammary development is usually symptom free. The patient with irregular menstruation, polymenorrhea or oligomenorrhea often has no pain The fertility quotient of the dysmenorrheic patient is statistically higher than that of her nondysmenorrheic sister.

The dysmenorrheic patient more often than not has normal cyclic menstruation, normal secondary sex characters and normal reproduction potentials. There is good clinical and laboratory evidence to support the opinion that the dysmenorrheic patient is endocrinologically normal. Endometrial biopsy at the onset of painful menstruation almost invariably shows a well developed secretory phase; it usually shows a secretory phase of the ++++ type. This is presumptive evidence of adequate or more than adequate progesterone secretion from the corpus luteum. The well developed progestational endometrium indicates a tissue capable of responding in prenidatory fashion to the estrogen and progesterone secretion of a corpus luteum. It implies a healthy, actively functioning corpus luteum, which in turn suggests that ovulation has occurred. For ovulation to have occurred, adequate gonadotropin from the anterior pituitary body must have been secreted to initiate and maintain this physiologic chain. It is obvious, then, that ovulation is an essential precursor to dysmenorrhea. Painless menstruation may be induced at will in the dysmenorrheic patient by inhibition of ovula-

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tion. Functional uterine bleeding of the menopause, which is usually anovulatory, is painless. From all accumulated evidence the patient with primary dysmenorrhea may be assumed to be endocrinologically normal.

Physiology of the Myometrium in Normal and Painful Menstruation

Usually there is no gross anatomic or histologic difference between the painfully menstruating uterus and the one that menstruates painlessly. The dysmenorrheic myometrium differs from the normal only in its pattern of contractility. The period of estrogen dominance during the first two weeks of the menstrual cycle induces a state of myometrial hypertonicity with small superimposed contractions. Under the influence of progesterone and estrogen secreted by the corpus luteum during the latter half of the cycle myometrial tonus is abolished, the contractions are increased in amplitude, and the contractile pattern is more rhythmic.

In the dysmenorrheic patient these orderly dynamics are altered.[1] The high-amplitude contractions of corpus-luteum influence are present, but they are superimposed upon a hypertonic uterus in which the intra-uterine pressure averages some 190 mm. of mercury. The intra-uterine pressure of the normal, nonpainful menstruating uterus is approximately 60 mm. The contraction waves are often "notched" and arrhythmic. Altered dynamics impair the expulsive efficiency of the uterus, and irregular shedding of the endometrium follows. Normally, the endometrial débris is expelled by a gradient of intra-uterine pressure from fundus to cervix. Pressure is highest at the fundus and lowest at the cervix. The differential is increased during the height of contraction. In the dysmenorrheic uterus this orderly pressure sequence is not found. The pressure at the internal os may actually exceed that of the fundus even at the peak of a contraction wave.[2] It may be assumed that dysmenorrhea is a syndrome associated with hypertonic, dysrhythmic contractions in which there is a reversal of the pressure gradient within the uterine cavity. This altered pattern of uterine contractions reflects other, more subtle physiologic changes. It is now generally agreed that the menstrual phenomenon is basically a vascular syndrome. Vasoconstriction in the endometrium induced by estrogen-progesterone withdrawal, spiral end-artery constriction and catabolic changes in the endometrium are the essential physiologic precursors to menstruation. Evidence is accumulating that the vascular bed is responsible in dysmenorrhea. The disordered mechanics of the myometrium just described may be the result of vasoconstriction, the smooth-muscle spasm being the sequel to oxygen deficit. Time-honored treatments for dysmenorrhea are alcohol, a vasodilator, and heat, a vasodilator. The most effective drugs against primary dysmenorrhea have a sympathicolytic action.

Relief After Hypogastric-Nerve Resection

Hypogastric-nerve resection relieves the pain of dysmenorrhea, at least in part, through its vasodilating effect. Pregnancy may relieve the pain of menstruation through the same mechanism. In a simple experiment previously reported the hypogastric plexus was exposed in a patient in whom an intra-uterine balloon had previously been inserted.[3] Stimulation of the plexus by means of a low-voltage current produced vasoconstriction as determined by pallor of the uterus and altered patterns of uterine contraction of the dysmenorrheic type as shown by balloon tracings. Upon division of the plexus and stimulation of the distal end, flushing of the uterus occurred, and this was accompanied by a drop in tonus and a slow rhythmic pattern of contractility. Further evidence of the relation of dysmenorrhea to vasoconstriction is to be inferred from the work of the Smiths.[4-7] An irritating globulin is said to be produced in the endometrium during the first hours of menstruation in amounts proportional to the amount of tissue destruction.

Dysmenorrhea is seen only in cases in which tissue destruction is high — namely, menstruation from a well developed secretory endometrium. Here it may be postulated that the amount of irritating globulin is high. The pain of dysmenorrhea can be relieved by blocking of the sympathetic pathways to the uterus without demonstrable effect on the sensory mechanism of other organs supplied over the pathways. This suggests that the relief following presacral neurectomy is not entirely the result of division of the afferent fibers. At least two physiologic changes that favor painless menstruation must be induced within the uterus. In the first place, the vascular dilatation in the myometrium that follows sympathectomy favors smooth-muscle relaxation. Secondly, the parasympathetics, which become dominant after presacral neurectomy, induce a rhythmic pattern of motility that favors expulsion of the endometrial débris and its "menstrual toxin."

Experimental Induction of Dysmenorrhea

On the theory that all the clinical manifestations of dysmenorrhea could be induced by vasoconstriction within the myometrium an attempt was made to reproduce the syndrome by vasoconstrictor drugs. The most potent of the vasoconstrictors is Pitressin, the vasopressor factor from the posterior lobe of the pituitary body. It has been shown that several of the clinical aspects of the dysmenorrheic syndrome could be produced by the intravenous administration of aqueous Pitressin. The intravenous administration of 5 units of Pitressin to the dysmenorrheic patient during the few days

prior to menstruation is invariably followed by typical intermittent contraction pain of dysmenorrhea. Furthermore, the regular pattern of uterine contraction assumes the hypertonic pattern in no way different from that which appears spontaneously in association with dysmenorrhea. Parenthetically, other symptoms commonly associated with dysmenorrhea such as headache, abdominal fullness and a positive water balance can be reproduced by Pitressin. The intravenous administration of Pitressin to the nondysmenorrheic patient is not followed by such noteworthy symptoms. It would be easy to postulate that the entire symptom complex of dysmenorrhea is the result of increased circulating Pitressin. However, this is not true. The blood plasma of the dysmenorrheic patient does not carry any measurable amount of vasopressor substance. It can only be said that the clinical syndrome of dysmenorrhea can be reproduced in part by the administration of a vasopressor substance. It may be that the dysmenorrheic uterus reacts abnormally to vasoconstrictor substances. Perhaps the "menstrual toxin" of the Smiths present in abnormally large amounts sensitizes the vascular bed of the myometrium to react in exaggerated fashion to the physiologic stimuli of menstruation. Since clinical dysmenorrhea can be reproduced by Pitressin it follows that treatment might be directed toward desensitizing the vascular response to the end that vascular constriction in the myometrium during menstruation is minimized.

Pitressin in the Treatment of Dysmenorrhea

It has long been known in the laboratory that repeated injections of Pitressin to animals over a period of days is followed by a progressive loss of vascular response to the hormone. Hypertension, which invariably follows the administration of Pitressin, becomes less and less after each subsequent injection. The long-continued administration over a period of days is followed by a complete inability of the peripheral or splanchnic vascular bed to react to vasopressin.[8] It is assumed that this elevated threshold to Pitressin is the result of desensitization of the vascular bed rather than to the development of antihormones. It is not necessary to go into the evidence to support this statement, but it is reasonably convincing.

It was thought that this laboratory observation might be utilized clinically in the treatment of patients with primary dysmenorrhea. If the vascular response to vasopressin could be reduced by repeated administration such desensitization to Pitressin might make the vascular bed of the myometrium likewise nonresponsive to the physiologically induced vasoconstrictor of menstruation.

It has been shown that the experimental production of dysmenorrhea by Pitressin was best accomplished during the few days immediately preceding expected menstruation. During the first half of the cycle no uterine spasm could be induced because the corpus-luteum hormone is essential for vascular response to Pitressin in the human being. On the basis of this information an attempt was made on 19 patients with severe primary dysmenorrhea to desensitize their vascular systems to Pitressin. In the first few patients the drug was administered during the last fourteen days of the cycle starting with 0.5 unit of aqueous solution and building the dose up to the patient's tolerance. This plan of treatment had several serious drawbacks. It required daily visits for injection and the response to the aqueous Pitressin in the form of headache, intestinal cramps, uterine cramps and other side effects was often quite unpleasant.

Sixteen patients have been treated with Pitressin in oil by a schedule that is now believed to be followed by maximum desensitization. In tests upon the blood-pressure response in rabbits it was shown that the maximum loss of vascular response occurs after five injections of Pitressin in oil given every other day. Experience has demonstrated that injection of 10 units of Pitressin in oil every other day for five doses approaches the optimal dosage schedule in the human being. The last 16 patients have received 10 units of Pitressin in oil on the fifteenth day of the menstrual cycle, and the same dose every other day until the onset of menstruation. Complete symptomatic relief of pain has been obtained in a relatively high percentage of the patients so treated. The longest follow-up study is on 2 patients who have gone one year since receiving their course of therapy. Both have remained asymptomatic. Eight patients have been completely free of pain for six months or longer since completing treatment. There were 3 patients in whom clinical relief of symptoms was unsatisfactory, analgesics being required for relief of menstrual pain. In 2 patients results were unsatisfactory, and subsequent operation disclosed pelvic endometriosis. The prodromal symptoms of menstruation, such as headache, breast fullness and backache, were significantly improved in all 16 patients.

The administration of Pitressin in oil is followed by no unpleasant reactions. Furthermore, the slow absorption favors desensitization of the vascular bed to subsequent injections of the vasopressin.

It seems that in severe primary dysmenorrhea this method of treatment might be worthy of further study.

Conclusions

Primary dysmenorrhea is a clinical syndrome characterized by two groups of symptoms: those arising from uterine muscle dysfunction — namely, hypertonicity, dysrhythmia and inverted pressure gradients interfering with expulsion of endometrial débris; and those related to altered electrolyte metabolism and water retention.

It is possible experimentally to induce the clinical symptoms of dysmenorrhea, both those arising from myometrial dyskinesia and those arising from positive water balance, by administration of the vasoconstrictor hormone, Pitressin.

The vascular response to this hormone may be reduced by repeated injections, through either the development of antihormones or desensitization of the vascular bed.

This effect has been employed to reduce the vascular response to the endogenously produced vasoconstrictor of menstruation, thus relieving the symptoms of dysmenorrhea.

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MEDICAL PROGRESS

DIABETIC COMA

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THOUGH there have been many articles on the therapy of diabetic coma, few have reviewed the available data pertaining to the etiology and quantitative definition of the extracellular and intracellular losses sustained by patients suffering the starvation and dehydration of coma. Such a review together with a discussion of the implications of these losses to parenteral therapy is the principal purpose of this communication.

Endocrine Factors

Inability to utilize glucose to satisfy energy requirements is the primary cause of the metabolic disturbances that result in the losses sustained by the patient in diabetic coma. That deficiency of insulin results in decreased utilization of glucose is substantiated by the effect of total experimental and therapeutic pancreatectomy and of destruction of pancreatic islet tissue, as indicated by histology or insulin assay after administration of anterior pituitary extract[1-4] or alloxan.[5, 6] The importance of insulin in this metabolic defect is also clearly established by the effect of insulin therapy.

On the other hand, animal experiments have shown that the anti-insulin factor of the anterior pituitary body[1, 4, 7, 8] or the antianabolic or catabolic hormones of the adrenal cortex[9-11] are diabetogenic. Observations on patients with acromegaly[12, 13] and adrenal tumors[13, 14] and on the effects of pituitary adrenocorticotrophin,[15-17] adrenocortical hormones[17-19] and stimulation of the hypothalamic-pituitary-adrenal axis[20] by the injection of epinephrine[21] or the alarm reaction[22] demonstrate that factors other than insulin diminish glucose utilization, increase oxidation of fat and tissue protein and favor hyperglycemia and ketosis.[23] Little is known of the clinical significance of tissue insulinase[24] or the hyperglycemic-glycogenolytic factor, suggested to be of pancreatic-acinar[1] or alpha-cell origin,[25, 26] which is found in higher concentration in most United States commercial insulin than in Danish insulin. Of interest are the observations that from 90 to 95 per cent of the pancreas must be removed to produce diabetes in the dog,[27] that such removal or alloxan causes severer diabetes than total pancreatectomy[6] and that pancreatectomy on a pituitary-diabetic or alloxan-diabetic dog may lessen the insulin requirements.[1, 5, 6] Yet the pituitary-diabetic and alloxan-diabetic dogs live longer and have less ketosis on withdrawal of insulin than pancreatectomized dogs.[1, 6] Without these antiinsulin or hyperglycemia factors the greater insulin requirement of diabetic persons or animals than that of those who have suffered total pancreatectomy would be puzzling.[1, 5, 6, 28, 29]

The role of hexokinase[30-32] and doubt regarding altered hexokinase activity providing the explanation of the disordered glucose metabolism in diabetes[33-35] have been reviewed recently in the *Journal*.[36] There reference also is made to the current work[37, 38] that points to insulin's action being dependent upon a chemical combination with tissue that may be inhibited by the inhibitors just mentioned.

But whatever the manner of insulin's role in utilizing glucose may be, diabetic coma appears to result from an intensification of the characteristic diabetic disturbance in the endocrine equilibrium

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between the secretion of the beta cells of the pancreas on the one hand and of the anterior pituitary, adrenal and thyroid glands on the other.[8, 23] Since the hyperglycemic factors other than insulin lack appear to play at times of infection, trauma and emotional stress an important role in the diabetic patient's increased need of insulin and extracellular and intracellular losses, their significance in the etiology, prevention and therapy of diabetic coma deserves more consideration than is frequently accorded them.[23, 39, 40]

Clinical Appraisal of the Patient

A carefully taken history will usually suggest the precipitating cause of the coma, the duration and severity of the metabolic disturbance, the resulting deficiencies and the extent of the therapeutic difficulties that may be encountered. The physical examination permits not only further appraisal of the patient's nutritional depletion, dehydration and acidosis but also detection of existing infection or acute abdominal or other complicating conditions.

The initial laboratory data still further define the immediate state of the patient. Blood and urine glucose and urine acetone and diacetic acid concentrations are essential in verifying the diagnosis. The ketonuria is also an index of the degree of starvation and excessive production of ketone acids in the body's unsuccessful attempt to meet caloric requirements by an acceleration of tissue catabolism.[41, 42] The degree of hyperglycemia, however, may not correlate with the degree of depletion, acidosis or seriousness of the patient's condition.[43, 44] Care should be taken to differentiate diacetic acid from salicylates, since patients suffering toxicity from the latter may present a picture occasionally mistaken for diabetic acidosis.[45] Blood hemoglobin and serum pH, protein, potassium and inorganic phosphorus concentrations are as important therapeutic guides as blood sugar, nonprotein nitrogen and serum carbon dioxide, chloride and sodium concentrations.[43, 44, 46-49] Hemoglobin or serum protein concentration may reflect hemoconcentration or, if not elevated in spite of the dehydration, the need of transfusion during therapy. The height of the nonprotein nitrogen, serum inorganic phosphorus or potassium indicates limitation of renal function imposed by reduced renal blood flow[50-52] in excreting the excessive load of tissue catabolites[42, 49, 53, 54] resulting from the starvation and dehydration characteristic of diabetic acidosis.[53-56] Information concerning the serum inorganic phosphorus and potassium concentrations is particularly important in view of the therapeutic considerations mentioned below. Determination by spectrographic flame photometry, when available, provides a rapid means of measuring serum potassium concentrations.[49, 57-59] The electrocardiogram permits immediate and repeated observations of the effects of disturbances in potassium metabolism on myocardial function and thus detection of possible potassium toxicity*[60-64] at the beginning of therapy[47, 49, 55, 64, 65] or of potassium deficiency† during therapy.[64-70] Parenthetically, it seems appropriate to comment that these toxic manifestations may not be dependent upon serum potassium concentration[71-73] but rather upon alterations in intracellular potassium or sodium concentration, ratio of potassium to sodium or release or pre-emptying of potassium in relation to glycolysis and glycogenesis.[74]

A single determination of the concentration of hemoglobin or serum protein is not a reliable index of the degree of dehydration without knowledge of the pre-dehydration level.[75, 76] Nor can concentration measurements of blood and plasma constituents without appropriate determination of volume be used to estimate losses sustained and therapeutic replacement needs.[77] An eosinophil count may provide an index of the role that hyperactivity of the adrenal cortex is playing in the tissue catabolism, renal excretion and insulin resistance.[16, 21, 23, 39, 78]

The serum CO_2 (carbon dioxide) concentration per se is less helpful to the physician than is generally assumed. Rarely does it contribute to the diagnosis. Unless the physician knows whether the patient is suffering from a metabolic, a respiratory or a central-nervous-system disturbance, the CO_2 concentration may not distinguish between acidosis and alkalosis.[43, 75, 76, 79-81] If the diagnosis is clear, as is usual in diabetic coma, the hyperpnea indicates a metabolic acidosis with a serum CO_2 of less than 13 milliequiv. per liter (29 vol. per cent). Its exact value is not necessarily an indication of the degree of acidosis or the seriousness of the patient's condition.[43, 44, 48, 81-83] A patient in such a state of shock that he is not responding to acidosis by hyperventilation may have a severer acidosis with lower serum pH but higher CO_2 concentration than the patient whose serum pH level is being defended by vigorous hyperpnea lowering alveolar CO_2 and serum H_2CO_3.‡[43,81] The misleading nature of a serum CO_2 concentration is nicely illustrated in patients given "alkali" as calculated by certain formulas to correct rapidly the serum bicarbonate deficit. Such patients may suffer an alkalosis with abnormally high pH before the CO_2 concentration has reached normal, possibly because a sluggish response of the respiratory center in lessening the hyperpnea delays elevation of the alveolar CO_2 tension and serum H_2CO_3.[43, 44, 80, 81] In other words, the patient's severe *metabolic acidosis* rapidly changes to a *respira-*

*Electrocardiographic changes characteristic of potassium toxicity are: peaking of the T wave, depression of the ST segment, absence of the P wave and widening of the QRS complex.

†Electrocardiographic changes indicative of potassium deficiency are: lowering, rounding and lengthening or inversion of the T wave, depression of the ST segment, prolongation of the QT interval and low voltage of the QRS complex. In the presence of a rapid heart rate, prolonged QT interval due to potassium deficiency and peaked T wave incident to acidosis, the T and P waves may coincide and give a combined wave that masks the T wave changes characteristic of potassium deficiency.

‡Alveolar $CO_2 \equiv$ serum H_2CO_3 and $pH \equiv \frac{BHCO_3}{H_2CO_3}$

tory alkalosis[75, 79-81] — a result that may be inconsistent with sound therapy providing an orderly restitution of the normal physiologic state.

Because neither the hyperpnea nor serum CO_2 may reflect quantitatively the degree of the acidosis either initially or later in the course of treatment, determination of serum pH is far more informative than determination of serum CO_2.

After the initial appraisal, careful clinical observations of the individual patient's response from hour to hour, supplemented by such laboratory data as are indicated, provide the only satisfactory criteria by which therapy can be directed promptly to meet specific needs according to the patient's changing condition.

Extracellular and Intracellular Disturbances

The starvation and dehydration incident to diabetic coma appear to result in a loss of potassium from the body that exceeds the catabolism of tissue as represented by nitrogen loss.[53-56] In the early phase of such starvation a portion of this excess of potassium over nitrogen loss appears to represent utilization and depletion of body glycogen.[84] Later, the loss of potassium is largely from cells,[56] coincident with the dehydration[85-88] and possibly acidosis.[43, 44] This loss from cells may be augmented by the administration of physiologic saline solution[89] or even hypotonic sodium chloride,[90] sodium going into the base-depleted cells[87, 88, 91-94] while the serum chloride rises or the urinary excretion of chloride increases.[95-97] Administration of an alkaline sodium salt appears to favor this passage of sodium to cells.[98]

Review of past therapy in diabetic coma reveals that infusion in the first eight to eighteen hours of therapy of liberal amounts* of so-called physiologic saline solution,† [96, 99] supplemented by large amounts of sodium bicarbonate or sodium lactate[81, 100-105] have provided in these hours of therapy‡ 15 or more milliequiv. of sodium per kilogram of body weight as compared to the approximately 7 milliequiv. per kilogram that would appear to cover the immediately replaceable losses incurred by the starvation and dehydration incident to coma during this short period of therapy.[53-55]

Such therapy seems to derive from concepts that do not necessarily apply to diabetic acidosis. In the first place, isotonic saline solution is far from physiologic.† Next, bicarbonate deficit as measured by serum CO_2 concentration may not be an index of sodium need. So far as the diminished CO_2 concentration reflects accumulation of ketone and other acid catabolites in the plasma, the deficit is dependent upon added acid rather than sodium loss. Moreover, since these acid catabolites are removed by oxidation and renal excretion in the course of insulin and repair therapy, plasma bicarbonate may be increased without addition of "alkali."[53-55, 76, 77, 106] In what appears to be the first attempt to calculate the amount of sodium bicarbonate§ that should be given to raise a low bicarbonate concentration to a normal value,[100] the assumption was made that bicarbonate was equally distributed throughout the body fluids. Data were then obtained on a mixed group of patients with cardiac disease, nephritis and diabetes that were interpreted as supporting the assumption. Thus, a faulty assumption was supported by data that ignored the differences in oxidation and excretion of the retained acid catabolites in the metabolic disturbances characteristic of each type of patient. Others‖[101-105] failed to take adequate account of the different concentrations of bicarbonate in extracellular and intracellular fluids. Because the shift in intracellular sodium with acidosis and dehydration has not been clearly enough defined to calculate the amount of sodium required to restore the normal intracellular concentration of approximately 8 milliequiv. of sodium per liter of cell water,[93, 94, 107, 108] modifications of the formulas used¶ are neither theoretically correct nor therapeutically reliable.[43, 44, 55, 80, 81, 106] Oddly and unfortunately, metabolic-balance data that determine the ultimate disposition of the large amounts of sodium lactate or bicarbonate used are not available.

A syndrome of peripheral muscle paralysis, respiratory muscle weakness, with shallow, gasping respiration, and myocardial failure, with weak, rapid pulse and ashen gray pallor, has been observed [49, 55, 65, 67-70, 109-112] in the course of parenteral rehydration of persons who have suffered intracellular depletion. It has been noted in several clinics that death may occur after the acidosis and the serum sodium, chloride and glucose concentrations had been corrected and in the absence of demonstrable cause at post-mortem examination.[68, 69, 81, 103, 111-114] That such deaths result, as suggested some years ago[115] from a therapy focused on extracellular fluid concentrations at the expense of intracellular needs, seems well substantiated by the prompt benefit of potassium therapy[67, 68, 110, 112] and by the metabolic data now available.[53-56, 70, 97, 112] The correlation between severity and duration of the acidosis and mortality in diabetic coma may be dependent upon acidosis inhibiting cellular syntheses with a loss of potassium, magnesium and phos-

*60-100 cc. per kilogram of body weight in eight to eighteen hours which equals 9 to 15 milliequiv. of sodium per kilogram.

†This solution is physiologic only so far as it does not cause shrinkage or increase in size of red cells and has approximately 150 milliequiv. of sodium per liter, as compared to the 140 milliequiv. of plasma. But it is not physiologic in its 150 milliequiv. of chloride as compared to the 105 milliequiv. of plasma, and it is not physiologic in terms of providing water for renal excretion and extrarenal loss and water and electrolyte for repair of dehydrated cells.

‡60-90 cc. per kilogram of body weight in eight to eighteen hours, which equals 10 to 15 milliequiv. of sodium per kilogram.

§1 gm. per kilogram of body weight equals 38 vol. per cent rise in blood CO_2.[44, 100, 103]

‖Milliequivalents of sodium needed = $\frac{60 - \text{vol. per cent serum } CO_2 \text{ found}}{2.24}$ x 0.7 kg. of body weight.[101, 103, 104]

¶Cubic centimeter of one-sixth molar lactate or bicarbonate needed = 55 − vol. per serum CO_2 x weight in pounds.[105]

phorus from cells,[43, 44, 116] the loss of these elements then being a limiting factor in essential syntheses[117, 118] and utilization of glucose.[119]

Definition of Losses Sustained

As early as 1924 the ineffectiveness of insulin alone in the treatment of dogs in diabetic coma was demonstrated.[120] Conversely, the beneficial effect of saline infusions has been shown by clinical experience and animal experiments to be but temporary unless insulin is given. Though glucose infusion with hyperglycemia may augment the body's utilization of glucose,[121-123] the increase is inadequate to meet the metabolic needs.

Since the losses incident to diabetic coma cannot be readily determined at the time the patient is seen and found to be in need of immediate replacement therapy, their appraisal demands a quantitative understanding of the metabolic losses sustained by the average patient in coma. In addition therapy must include the maintenance requirements of the patient during the period of replacement therapy.[55] Hence, a quantitative knowledge of such losses and requirements is basic in the treatment of diabetic coma. If the data are at hand, therapy providing water, specific electrolytes and calories in amounts conforming to repair and maintenance needs may be promptly administered in a physiologic manner.

This section, therefore, reviews data that permit quantitative estimation of such losses and therapeutic needs.

The marked depletion of tissue glycogen incident to diabetic coma was demonstrated in 1917.[124] The deficit of glycogen has been estimated as more than 3 gm. per kilogram of body weight.[125]

The increasing plasma concentrations of glucose, ketone acids and other catabolites result in a several-fold increase in solute load presenting for renal excretion,[54] which commands an increase in minimal urine volume.[126] Moreover, the limitation in urine concentration observed during the dehydration of diabetic acidosis[54] results in a renal water loss greater than demanded by a maximal urine concentration of 1.5 millimols per liter.[126] Such a limitation in urine concentration with increasing volume has been observed in short-time-loading experiments.[127-129] In these experiments the increase in osmolarity increases plasma volume by drawing water from cells and results in a diuresis, with an increased loss of sodium and chloride. In the diabetic person the polydipsia, shift of water from cells and loss of sodium and chloride in urine frequently result in a fall in serum sodium and chloride concentrations. The dilution of these relatively impermeable extracellular ions may then play a part in the diuresis by inhibiting the hypothalamic osmoreceptor's activation of antidiuretic-hormone production.[130] The continuing hyperglycemia may have little osmotic effect on the receptors and posterior pituitary hormone secretion, as do the permeable ketones and urea.[130] Or the adrenal steroids released by the alarm reaction may inactivate the antidiuretic hormone. But, whatever the explanation, the compensatory polydipsia does not keep abreast of the renal water loss, and a progressive dehydration occurs,[53, 54] the rate perhaps depending upon the degree of the alarm reaction incident to the illness, injury or emotional state of the patient. This dehydration, associated with a disturbance of normal cellular glycolysis, hyperglycemia and shift in water from cells to plasma,[131, 132] is characterized by cellular hydropenia with relatively little decrease in extracellular water until nausea and vomiting impose thirsting as well as starvation.[53-56] It is then that the polyuria due to the factors outlined above produces a precipitous dehydration.

The metabolic data mentioned[53-56] demonstrate that patients going into diabetic coma lose not only the water, chloride and sodium of plasma and extracellular fluid but also the water, phosphorus, sulfur, potassium and magnesium of tissue cells. The experiments on patients, however, were stopped too soon to reveal the magnitude of the total losses sustained in severe diabetic coma. In the absence of such metabolic data approximations concerning these losses are excusable and useful.

Table 1 presents in summary form estimations of dehydration losses per kilogram of body weight derived in different ways.[55] The first line presents purely theoretical calculations of losses incident to a dehydration resulting in a 10 per cent reduction in body weight, assuming extracellular water as 20 per cent, intracellular 45 per cent and total body water as 65 per cent of body weight,[133] an equal loss of extracellular and intracellular body fluids and no change in body-fluid concentrations. Such dehydration means reduction of approximately 17 per cent in total body water, 25 per cent in extracellular water and 12 per cent in intracellular water. The second line gives the actually determined losses (not including insensible losses) of electrolytes suffered by a normal adult during four days of complete thirsting and starvation.[55] The third line presents similar losses suffered by a diabetic patient during withdrawal of insulin for three and four-tenths days with occurrence of pre-coma acidosis and nausea as well as extrapolation of losses for an added day of vomiting and thirsting.[54, 55] If the diabetic person, as available data suggest,[54, 55] does not concentrate urine to the normal high value of 1.4 millimols per cubic centimeter,[126] the water loss of the added extrapolated day may be too small.

Since recent work[134] indicates concentrations of sodium, chloride or potassium of no more than 10 milliequiv. per liter of insensible water loss, the extrarenal loss of electrolytes appears small in the absence of appreciable sweating.

The loss of 3 milliequiv. of potassium per kilogram of body weight by the thirsting subject (second line of Table 2), as compared to the 7 milliequiv., derived by calculation (first line), appears to reflect the concentration of cellular potassium that occurs during the dehydration of thirsting. There is, however, an excessive loss of potassium from cells in relation to nitrogen, since the ratio of milliequivalents of potassium lost to grams of nitrogen lost (second line) is 4.3 compared to the ratio of approximately 2.8 of muscle tissue. This excessive loss of potassium above nitrogen from tissue catabolism appears characteristic of starvation and dehydration.[55, 85-88, 93, 94]

Nitrogen-balance data[55] suggest that a diabetic person ingesting food and water suffers more starvation when insulin is withdrawn than a normal subject does during complete starvation and thirsting. This apparently reflects the diabetic patient's inability to utilize the carbohydrate derived from tissue catabolism. In addition, the data of the third line in Table 1 indicate that the loss of potassium above nitrogen is striking in diabetic coma, the ratio of the losses being 6.6. That this nonprotoplasmic loss of potassium is not an artefact of the extrapolated theoretical day of thirsting included in the data is shown by the fact that the ratio is 6.8 for the first three and four-tenths days of actual metabolic balance.[54, 55] It appears to reflect the disturbed carbohydrate metabolism and the excessive intracellular dehydration to which reference has already been made. Under such circumstance reinstitution of the glycolytic cycle and glyconeogenesis and rehydration of cells to restore normal nitrogen concentration would result in a deficit of intracellular potassium, unless potassium were made available in the rehydration therapy. While the ratio of magnesium loss to nitrogen loss also exceeds the magnesium-nitrogen ratio of protoplasm, there is not such excessive loss of phosphorus over nitrogen.[53-56]

Despite the inaccuracies in these quantitative estimations of losses, the agreement of the differently derived data is somewhat reassuring. Babies dehydrated by diarrhea have been found to retain during several days of parenteral rehydration approximately 125 cc. of water, 9.5 milliequiv. of sodium, 9 milliequiv. of chloride and 10 milliequiv. of potassium per kilogram of body weight.[88] Per 100 cc. of water retained these figures are 7.6 milliequiv. of sodium, 7.2 milliequiv. of chloride and 8 milliequiv. of potassium. Comparable potassium retentions have been observed in patients during recovery from diabetic coma,[96] at least 3 milliequiv. per kilogram of body weight being required to prevent deficiency.[97] The maximal need for sodium has been estimated as 12 milliequiv. per kilogram.[91] Retentions during such therapy may overestimate actual hydration needs, since they may exceed losses[98] or reflect deposition of new tissue. The administration of physiologic saline solution without potassium has been shown to result in negative potassium balances, overexpansion of extracellular volume and such retentions of sodium and chloride that the balances of these two ions were negative during the subsequent periods when potassium was administered.[96]

TABLE 1. *Estimations by Several Methods* of Acute Losses of Water and Electrolytes per Kilogram of Body Weight, Assuming the Weight Loss Reflects Dehydration.*

Method*	Water Loss	Sodium Loss	Chloride Loss	Potassium Loss	Magnesium Loss	Phosphorus Loss†	Nitrogen Loss
	cc.	*milliequiv.*	*milliequiv.*	*milliequiv.*	*milliequiv.*	*milliequiv.*	*gm.*
1	100	7	6	7	—	—	—
2	90	5	4	3	—	3.5	0.7
3	100	5	4	6	0.8	4.0	0.9

*1 = calculation assuming 10 per cent weight loss, an equal loss from extracellular and intracellular fluids and maintenance of normal body-fluid composition.[55, 125]
2 = losses suffered during 4 days of complete thirsting and starvation.[55]
3 = losses suffered by a diabetic patient during withdrawal of insulin for 3.4 days plus added theoretical day of vomiting and thirsting.[54, 55]
†Mg. of phosphorus ÷ 31 = millimols; millimols x 1.8 = milliequiv. at pH 7.4.

It should be noted that the average ratio of sodium loss to chloride loss of 1.2 (Table 1) approximates that of extracellular fluid and thus, as frequently stressed,[53-55, 76, 77] indicates a loss of sodium above chloride that accords with loss of extracellular fluid. Physiologic repair of the extracellular sodium deficit would thus appear to be satisfied by the administration of sodium and chloride in approximately the ratio, not concentrations, pertaining to extracellular fluid rather than by the addition of "alkali" per se according to calculations that may entail such errors as those mentioned above.

Therapy

Insulin

A primary therapeutic consideration is the provision of adequate insulin. It is essential to the cessation of the starvation by re-establishing normal cellular carbohydrate metabolism upon which transfer of water and electrolytes from extracellular fluid to the dehydrated cells is dependent.[132, 135]

Little mention, however, has been made of the use of protamine zinc insulin or the newer crystalline protamine insulin[136] in the therapy of coma.

In this clinic we have routinely given with the first dose of crystalline insulin a separate injection of 5 to 40 units of protamine zinc insulin, depending upon the age, size and past history of the patient, including past therapy, if any. This insulin has little therapeutic effect in the emergency treatment, but it is believed to facilitate therapy later. It has never in our experience complicated therapy by hypoglycemia or in any other respect been found undesirable. The crystalline protamine preparation may prove preferable to the slower-acting protamine zinc insulin, particularly if the crystalline protamine is to be used in the subsequent routine insulin therapy.

The first dose of crystalline insulin of at least 2 units per kilogram of body weight or 75 units per square meter of body surface* [55, 137] should be given intramuscularly to avoid the possible delays of intravenous administration. A similar amount of crystalline insulin should be given intravenously with the first intravenous infusion. Subsequent need of crystalline insulin must be determined by the physician in continuous attendance on the patient after appraisal of the total information available from hour to hour. An order for insulin that is to be carried out an hour or more later is not a good therapeutic procedure.

To lessen possible insulin resistance, measurements should be taken to control the specific factors in each case that may evoke or continue an alarm reaction.[23, 39] Similarly, to increase the effectiveness of administered insulin, substances essential to the reinstitution of normal glycolysis should be provided.[44, 117-119] The recent observation that glutathione intravenously improves the diabetic patient's utilization of glucose[138] may ultimately be found to have therapeutic applicability.

Though diagnosis is not within the province of this review, one word seems justified. Because the hypoglycemia of severe insulin reactions may cause irreversible injury to cerebral cells,[139] insulin should never be given until hypoglycemia as the possible cause of the coma has been ruled out.

*Correlation of square meters of body surface with kilogram of body weight for a person of average configuration may be indicated as follows:
0.5 sq. m. of body surface = 11 kg. of body weight
1.0 sq. m. of body surface = 28 kg. of body weight
1.7 sq. m. of body surface = 65 kg. of body weight

Estimation of Replacement and Maintenance Therapy

In the application of such estimation of losses, as mentioned above, to replacement therapy consideration has been given the fact that the losses repairable by parenteral therapy do not include those referable to protoplasm lost from tissue oxidation.[55] The latter can be repaired only by the deposition of new tissue and fall outside the scope of the immediate rehydration therapy. Thus, replacement therapy of potassium, magnesium and phosphorus concerns only the losses that exceed the catabolism of tissue as measured by nitrogen balance. Moreover, since body water is proportional to body tissue, the rehydration required in the first twenty-four hours will be accomplished by less than 100 per cent replacement of water loss. Approximate repair requirements for the first twenty-four hours of therapy in terms of per cent of losses incurred have therefore been estimated[55] as water, sodium, and chloride, 80 per cent; potassium and magnesium, 50 per cent; phosphorus, 25 per cent; and nitrogen, 0 per cent (see Table 3).

During this twenty-four-hour period of repair therapy, the patient's maintenance requirements[55] must be provided in addition to the repair needs. These maintenance requirements, however, are prescribed by physiologic functions that are more nearly proportional to square meters of body surface than to kilogram of body weight.[55, 137] Their derivation is given in Table 2. Excessive glycosuria or continuation of acidosis or an alarm reaction may increase the requirements of water and electrolytes, particularly the intracellular electrolytes.

Table 3 presents a summation of immediate basic repair and maintenance needs per square meter of body surface during the first twenty-four hours of therapy as indicated by the considerations men-

TABLE 2. *Basic Parenteral Maintenance Requirements Per Square Meter of Body Surface of a Diabetic Patient during the First Twenty-Four Hours of Therapy.*

REQUIREMENT	WATER *cc.*	SODIUM *milliequiv.*	CHLORIDE *milliequiv.*	POTASSIUM *milliequiv.*	MAGNESIUM *milliequiv.*	PHOSPHORUS *milliequiv.*	GLUCOSE *gm.*
Insensible*	600	4	4	2	—	—	—
Renal:							
Normal basic†	250	6	6	10	3	12	—
Added diabetic‡	750	12	11	10	4	23	50
Caloric:							
Total	1600	22	21	22	7	35	300
Concentration per liter	1000	14	13	13	5	23	200

*Assuming that perspiration accounts for 70 per cent of insensible loss and has sodium and chloride concentrations of 10 per cent of the extracellular fluid concentration.

†Approximation by experimental data on normal subjects.

‡Approximation by metabolic balance data on patients in diabetic coma during therapy.

tioned. Because the repair needs reflect loss of a percentage of body mass, they are expressed per kilogram. The basic maintenance requirements, on the other hand, are expressed per square meter of body surface. The summation of the repair and maintenance data gives the total basic therapeutic requirements for a patient of 1 square meter. These, when reduced to a per-liter basis (last line of Table 3) have been used to define the magnitude of concentrations of electrolytes and glucose appropriate for parenteral fluid therapy.[55]

In view of the increasing evidence of the indispensable role of various ions in enzyme-catalyzed processes,[118] of the role of magnesium in the utilization of glucose,[140] of the tissue need of magnesium as indicated by balance studies and serum concentrations as low as 0.5 milliequiv. per liter during therapy[55, 106] and because of the lack of toxic symptoms until serum magnesium exceeds 6 milliequiv. per liter,[141] the provisions of 5 milliequiv. of magnesium per liter (Table 3) in the parenteral therapy appears appropriate.

The role of phosphorus in the glycolytic cycle and acid-base equilibrium[44, 142] and the occurrence of serum inorganic phosphorus levels of less than 0.6 milliequiv. per liter (or 1 mg. per 100 cc.) during the course of therapy indicate a particular need for this element,[143, 144] even though the balance data do not indicate loss of phosphorus in excess of nitrogen in terms of protoplasmic proportions.

The concentrations of sodium and chloride per liter suggested as appropriate for parenteral therapy (Table 3) indicate that so-called "physiologic saline solution" — that is, 150 milliequiv. of sodium and 150 milliequiv. of chloride per liter — is excessively hypertonic to plasma as regards chloride concentration. Also, in terms of the fluid requirements of cells, insensible water loss and renal excretion, it is hypertonic in both sodium and chloride. Indeed, the "physiologic" designation applies only to osmolar isotonicity and not to parenteral therapy. If sodium bicarbonate or sodium lactate were used to correct the serum CO_2 as frequently recommended and commented upon above, the total sodium administered in the first twelve hours of therapy might approximate several times the indicated twenty-four-hour need. If the water requirement were met by the administration of isotonic saline solution, approximately five times the indicated amount of chloride might be given.

However, a multiple electrolyte solution that has a composition per liter appropriate for the total needs over the first twenty-four hours should not be used in the initial parenteral fluid therapy. Too often, a patient's circulation and renal function are impaired. The serum potassium may be elevated to double and the inorganic phosphorus to triple the normal values.[17, 49, 55, 143] Under such circumstances administration of a solution containing 30 milliequiv. of potassium and 16 milliequiv. of phosphorus per liter might result in serious toxic effects.

On the other hand, use of isotonic saline solution whose hypertonicity in chloride ion may cause an elevation in serum chloride to 125 or more milliequiv. per liter[95, 96, 131] appears particularly unphysiologic for a patient suffering the exhausting hyperpnea of an acidosis so severe that the serum pH may be as low as 6.9.[43, 81] It has been suggested that the severity and duration of such an acidosis may determine not only continuation of intracellular losses[44] but also recovery.[43, 116] The decrease in respiratory minute volume, cerebral oxygen consumption and consciousness has been found to have an impressive correlation with arterial pH values below 7.[43] The custom in some clinics of correcting the acidifying effect of isotonic saline solution by the addition of molar sodium lactate or bicarbonate until the sodium concentration is elevated to 190 milliequiv. per liter in order to provide a sodium to chloride ratio similar to plasma — that is, 1.3 — results in a hypertonicity that does not accord with the need of water relatively free of sodium and chloride for cellular hydration, renal excretion and extrarenal loss. Similarly, reducing the chloride concentration of isotonic saline solution to the normal plasma value of 100 milliequiv. per liter by adding one part of one-sixth molar sodium lactate or bicarbonate to two parts of the

TABLE 3. *Repair and Maintenance Parenteral Therapy of First Twenty-Four Hours of Treatment for a Child of Approximately 30 kg. or 1 sq. m. of Body Surface.*

REQUIREMENT	WATER	SODIUM	CHLORIDE	POTASSIUM	MAGNESIUM	PHOSPHORUS	GLUCOSE
	cc.	*milliequiv.*	*milliequiv.*	*milliequiv.*	*milliequiv.*	*milliequiv.*	*gm.*
Repair* (per kg. x 30)	2400	145	120	90	12	30	0
Maintenance (per sq. m. x 1)	1600	22	21	22	7	35	300
Total	4000	167	141	112	19	65	300
Approximate concentration per liter	1000	42	35	30	5	16	75

*As 80 per cent of water, sodium and chloride losses, 50 per cent potassium and magnesium losses, 25 per cent of phosphorus loss; and 0 per cent of nitrogen loss, as stated in the text.

saline solution, as has been advocated,[76] appears to underestimate such needs for water. More recently[55, 106] hypotonic saline solutions to which enough sodium bicarbonate or lactate has been added to make the sodium to chloride ratio 1.3 have been used to provide initially not only water and electrolytes to improve plasma and extracellular fluid volumes but also water to meet cellular, insensible and renal water needs. Such a solution seems physiologically appropriate, provided an excessive rate of infusion does not cause hemolysis or further reduce an already low plasma sodium concentration.

It would be interesting to know if the oliguria that sometimes occurs in the treatment of coma is due to a shift of water from plasma to cells with the re-establishment of cellular glycolysis,[49, 129, 132, 135] to a diminished renal water requirement[126] or to cardiac failure incident to potassium deficiency.

Provision of Parenteral Fluid Therapy

Review in outline form of a generally accepted practice in the provision of parenteral therapy appears impossible since the therapy is divergent not only in different clinics but also for patients in a clinic. However, the considerations and repair and maintenance needs reviewed above perhaps suggest a parenteral fluid therapy in diabetic coma that may be outlined as follows:

1. Initial hydration is started immediately by the intravenous infusion of 600 to 800 cc. per square meter at 8 cc. (140 drops) per minute per square meter of a solution* containing 100 milliequiv. of sodium, 60 milliequiv. of chloride and 45 milliequiv. of bicarbonate or lactate per liter.[55, 91, 106] The passage of water to cells with the reinstitution of cellular glycolysis and from the body by insensible and renal loss of water counteracts dilution of extracellular sodium and chloride. The use of 5 per cent glucose for diluting isotonic saline solution or its addition to the initial parenteral fluids appears contraindicated, since it may result in an undesirable prolongation of hyperglycemia in some patients.[131] Glucose in initial therapy appears particularly undesirable when given in isotonic saline solution, for hyperglycemia may accentuate the limitation of such a solution in providing water to cells by increasing the osmotic gradient between extracellular and intracellular fluids. Its provision in a hypotonic solution may limit the deleterious effects by the hypotonicity of this solution in extracellular electrolytes increasing the availability of water to the hydropenic cells.

*One may make the solution readily by adding a 40-cc. ampoule of molar sodium bicarbonate or lactate and 550 cc. of pyrogen-free distilled water to approximately 400 cc. of isotonic saline solution (that is, 0.85 gm. of sodium chloride per 100 cc.).

2. After the improvement in circulation and renal function that usually occurs with the initial therapy, a whole-blood transfusion of 300 cc. per square meter is considered for the management of shock and deficits in hemoglobin and serum protein and is given if indicated. In some cases such a transfusion may be more appropriate later in the course of therapy.

3. After such initial hydration, if the patient is voiding and the circulation is satisfactory, a hypotonic multiple electrolyte solution such as indicated in the last line of Table 3, containing 2 to 5 per cent glucose, depending upon the hyperglycemia, is infused at a rate of approximately 3 or 4 cc. (50 drops) per minute per square meter.[55, 105] A low concentration of glucose appears preferable until the blood sugar has fallen to approximately 200 mg. per 100 cc.

4. As this infusion proceeds, the patient is carefully followed for indication of the need of:

a. Insulin, as reflected by changes in degree of glycosuria and ketonuria in frequently voided specimens or urine obtained directly from catheter each hour and by blood sugars as need is felt for obtaining accurate information concerning the adequacy of decrease in hyperglycemia or possibility of hypoglycemia. If the glycosuria and ketonuria are not improving, insulin should be given liberally. When the glycosuria is improving rapidly and the ketonuria has disappeared, insulin should be given with caution and in relation to carbohydrate intake. If the ketonuria persists after the glycosuria has ceased, the carbohydrate intake should be increased and insulin given as needed to control any recurring glycosuria.

b. Fluid, as indicated by continual reappraisal of dehydration and circulation and by estimation of water balance by intake and output.

c. Sodium and chloride to provide the extracellular electrolytes for the expanding extracellular volume and to replace continuing losses by urine or vomitus. Satisfaction of water requirements by some such multiple electrolyte solution as designated above will usually provide the required sodium and chloride, unless their loss continues abnormally.

d. Provision of potassium, as reflected by intake, symptoms, serial electrocardiograms and when possible serum potassium concentrations. The addition of 1-gm. ampoules of potassium chloride — that is, 15 milliequiv. of potassium — to the multiple electrolyte infusion, which is so convenient in providing requirements in the postoperative alkalotic patient,[145] may prolong the acidosis in the diabetic person by decreasing the ratio of sodium

to chloride. Hence, the potassium might better be added as potassium acetate or lactate or, if phosphorus is needed, as 7 parts of dipotassium phosphate and 1 part of monosodium phosphate. With the latter there would appear to be little danger in giving excess phosphorus so long as the serum inorganic phosphorus remains at or below normal levels.[143] Care should be taken to use highly purified salts. As long as indications of potassium deficiency are present, addition of potassium to raise the concentration of the parenteral solution to 60 milliequiv. of total potassium per liter may be desirable.[49, 112]

e. Phosphorus, as indicated by estimated need minus intake and by serum concentrations of inorganic phosphorus. Usually, the multiple electrolyte solution will meet the phosphorus needs until diluted milk can be given orally.

In most clinical laboratories there is no means of determining plasma magnesium concentrations readily enough to guide therapy even if need of accurate provision of this element should be demonstrated.

When blood samples are collected for the above determinations or when the patient's respirations or course causes concern, the serum pH and CO_2 should be determined.

5. The multiple electrolyte glucose solution is continued until fluid can be taken orally or the repair and maintenance needs are covered. The duration of parenteral therapy varies according to the persistence of nausea. Usually 70 per cent of the twenty-four-hour requirements given parenterally in twelve hours by the initial hydrating therapy, the transfusion and the multiple electrolyte therapy will result in such improvement that the remainder may be given orally. When the solution can be given orally, it may be flavored with gingerale or orange juice or diluted with milk.

After the blood sugar has begun to fall toward 200 mg. per 100 cc., the inclusion of glucose in the parenteral therapy in conjunction with insulin and other substances (potassium, magnesium and phosphorus) essential to re-establishment of the glycolytic cycle appears desirable. This provides a source of energy to check tissue catabolism and ketosis and a supply of glucose to replenish the depleted glycogen stores. With provision of the electrolytes favoring utilization of glucose the possible deleterious effects observed in giving glucose in isotonic saline solution at the inception of therapy[131] should be avoidable. Under the disturbed metabolic conditions pertaining in diabetic acidosis, use of the respiratory quotient as an index of glucose utilization seems unsound and appears to have underestimated[146] glucose utilization despite the presence of severe diabetic acidosis and the omission of potassium therapy.[131] Today, rather than giving no glucose to avoid increasing the need of intracellular substances such as potassium, magnesium and phosphorus,[74, 147] it appears more physiologic to provide these substances in amounts that permit the administration of such glucose as will meet the physiologic needs. Therapy should not prolong a hyperglycemia that inhibits intracellular hydration and continues the diuresis.[131] More metabolic data on patients receiving adequate insulin, glucose and the essential intracellular substances are needed for a better definition of optimal provision of exogenous glucose.

An even more hypotonic electrolyte solution than that suggested in Table 3 has been used as the multiple electrolyte parenteral therapy[91, 106]—namely, one containing approximately the following: 30 milliequiv. of sodium, 22 milliequiv. of chloride, 20 milliequiv. of potassium, 8 milliequiv. of phosphorus and 20 milliequiv. of lactate per liter and glucose as needed to satisfy the caloric needs. With such a solution for parenteral fluid therapy based on repair and maintenance needs as outlined above and administered with precautions to avoid toxicity from potassium,[55, 91, 106] good clinical results without evidences of potassium toxicity have been obtained in the rehydration of several hundred patients suffering dehydration with acidosis from diabetes,[55, 106] diarrhea[91, 148] or burns and also with alkalosis from vomiting or postoperative dehydration and alarm reaction.[145] More recently a multiple electrolyte solution has been used conforming more closely to that indicated by the data of Table 3 and containing approximately 55 milliequiv. of sodium, 45 milliequiv. of chloride, 23 milliequiv. of potassium, 5 milliequiv. of magnesium and 12 milliequiv. of phosphorus per liter and a pH of 7.4.*

Much larger amounts of potassium and phosphorus than those provided in the solution mentioned above have been administered[49, 112, 143] without abnormal elevation of serum concentrations. The readiness with which potassium is transferred from plasma to cells after the first two or three hours of initial therapy has been clearly demonstrated.[49] Solutions containing as much as 75 to 155 milliequiv. of potassium per liter have been infused without undue elevation in serum potassium or undesirable changes in the electrocardiogram,[49] but the rates of such infusions were not given. However, 40 to 90 milliequiv. of potassium per liter has been given intravenously to adults at 8 to 12 cc. per minute without toxic manifestations.[112]

*Made by the addition of 2.8 gm. of sodium lactate, 1.8 gm. of sodium chloride, 1.0 gm. of potassium chloride, 1.0 gm. of K_2HPO_4, 0.14 gm. of $NaH_2PO_4.H_2O$ and 0.25 gm. of magnesium chloride to 1 liter of 2 to 10 per cent glucose.

The sodium and potassium salts and 40 cc. of water are delivered to a small, 50-cc. flask, which is capped and autoclaved. The magnesium chloride and 20 cc. of water are delivered to a separate 30-cc. flask and autoclaved. (Autoclaving of the magnesium and phosphate salts in a single solution may result in cloudiness.) The contents of the two sterile flasks are added to a liter of sterile glucose solution (2 to 10 per cent glucose as indicated) for the multiple electrolyte-glucose infusion.

Such concentrated solutions of potassium should, however, be administered with caution and the taking of serial electrocardiograms since rapid administration may cause cardiac failure.[149] A recent report[97] on a sizable series of patients indicates that from 200 to 300 milliequiv. of potassium is required to meet the potassium deficit of the average adult patient in coma. This agrees well with the per kilogram loss of 3 to 4 milliequiv. of potassium in excess of nitrogen suggested by the metabolic data presented above.

Summary

Consideration of the contributions reviewed here appears to clarify certain aspects of therapy concerning which there has long been debate.

The unphysiologic nature of physiologic saline solution as repair and maintenance parenteral therapy is emphasized. It is not appropriate for prompt amelioration of the severe acidosis, repair of intracellular deficiencies and provision of water for insensible and renal water requirements.

The provision of sodium bicarbonate or sodium lactate as frequently done according to calculations based on plasma carbon dioxide deficit is neither theoretically correct nor practically desirable. It tends to sodium overdosage, producing alkalosis and distortion of intracellular composition that may predispose to serious and at times fatal sequelae.

In conjunction with insulin the provision of intracellular substances essential to the re-establishment of normal cellular composition and function should be a routine consideration in the provision of optimal hydration therapy. Appropriate provision of these intracellular substances and insulin usually enables the physician to administer, after the initial two hours of parenteral therapy, such glucose as is required to meet caloric needs and replenish glycogen stores. The provision of glucose in the initial parenteral fluid therapy or until the hyperglycemia is diminishing appears undesirable.

Thus, the metabolic data and clinical experience available today permit not only quantitative appraisal of repair and maintenance needs but also delineation of a program of therapy that may be modified to satisfy physiologic requirements and should avoid some of the complications and deaths that have occurred in the past. But, as is often the case, better definition of metabolic disturbances and of more physiologic therapy makes the physician's task more exacting as well as satisfying.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 36431

PRESENTATION OF CASE

A sixty-five-year-old man was admitted to the hospital because of pain in the neck and weakness of the right arm and shoulder.

Two years before entry, while working in the woods, he tripped over a log. He had amnesia for the fall and only remembered lying on the ground a few moments later. There were no contusions or lacerations; however, he noted a severe pain in the back of the neck. He was able to drive home but was immediately taken to a hospital. Radiologic examinations revealed a fracture of the second cervical vertebra. For one month he remained in the hospital, where he was maintained on constant head traction. However, when the traction was removed, he noted severe neck pain when sitting upright. He was therefore placed on head traction at home for a second month. Following this he still developed slight pain in the back of his neck when sitting upright but was able to hold a job as a night watchman until four months before admission. Approximately five months prior to entry there was weakness in the right shoulder, arm and hand, which gradually progressed until the right hand was almost useless. In recent weeks he also noted some weakness in the legs, more marked on the right, limitation in motion of the neck and a sensation of faintness when lying on the right side. One month before entry he developed a severe cough, which cleared up after a week of hospitalization.

Physical examination revealed a well nourished man in no acute distress. Examination of the heart, lungs and abdomen was negative. There was marked restriction in the mobility of the neck, especially in side-to-side motion, and marked weakness in raising the right shoulder. He could abduct the right arm only 35°, and the biceps and triceps were weak. There was no abduction or adduction and only weak apposition of the right fingers and thumb and atrophy of the musculature of the right hand, forearm and arm. He had sensory loss to pinprick over the left ulnar and radial distribution and poor position sense of the left hand. The Babinski reflex was positive, and there was sustained ankle clonus on the right side but none on the left.

The temperature was 98°F., and the pulse 80. The blood pressure was 145 systolic, 90 diastolic.

Examination of the blood revealed a hemoglobin of 14.8 gm. and a white-cell count of 9300, with 55 per cent neutrophils, 27 per cent lymphocytes, 7 per cent monocytes and 11 per cent eosinophils. A blood smear disclosed normal-appearing red cells and an increase in the number of platelets. The nonprotein nitrogen was 29 mg., the total protein 7.07 gm., with an albumin-globulin ratio of 1.1, the calcium 9.7 mg. and the phosphorus 3.0 mg. per 100 cc., the carbon dioxide 27.3 milliequiv. per liter and the prothrombin time 70 per cent of normal. A lumbar puncture disclosed an initial spinal-fluid pressure equivalent to 195 mm. of water and a final pressure equivalent to 160 mm. of water, with a rise on compression of the right and left jugular vein of not more than 5 mm. The spinal fluid was clear, with a red-cell count of 12 per cubic millimeter and a total protein of 160 mg. per 100 cc.

Radiologic examination of the chest revealed normal lung fields and slight cardiac enlargement. X-ray examination of the lumbodorsal spine and pelvis showed demineralization in excess for the age of this patient in all visualized bones and irregular sclerosis of the cortex of the left ischium and pubic bone. There was slight wedging of the bodies of the first and second lumbar and eleventh and twelfth thoracic vertebras. Radiologic examination of the skull and neck disclosed slight generalized demineralization. There was an irregular destructive lesion invading the body and pedicle of the third cervical vertebra (Fig. 1), the greater part of which appeared to have been resorbed with collapse of the remaining portions. This resulted in a sharp kyphosis of the upper cervical spine. There was bulging of the bone anteriorly, causing a prominence of the soft tissue of the nasopharynx. The cortex of the bone appeared to have been broken through in several points. There were osteolytic areas in the lamina and spinous processes of the third cervical vertebra. No other localized areas of bone destruction were seen.

On the third hospital day the third cervical vertebra was biopsied through the nasopharynx.

DIFFERENTIAL DIAGNOSIS

DR. JOHN A. REIDY*: This is the case of an elderly man who apparently sustained a fracture of the cervical spine two years before admission. Whether or not that was a pathologic fracture was not re-

*Assistant orthopedic surgeon, Massachusetts General Hospital.

corded. Nevertheless he made a fairly adequate recovery after two months' time and was able to go to work as a watchman. He did well except for some pain in the back for over a year, and then began to have weakness and difficulty in using the right arm and right leg. These signs were consistent with some compression of the nerve roots and spinal cord in the cervical region. The x-ray films were described as showing a destructive lesion of the third cervical vertebra, which was biopsied. Certainly,

FIGURE 1.

this is a difficult type of lesion to diagnose without a biopsy.

If Dr. Wyman will let me look at the x-ray films, they might be helpful.

DR. STANLEY M. WYMAN: The cervical spine (Fig. 1) shows this very sharp kyphos in its uppermost portion. The lower cervical spine has a normal curve, but beginning at approximately the level of the third cervical vertebra and going up to the first and second cervical vertebras there is a very sharp anterior angulation. The body of the third cervical vertebra, probably the second cervical vertebra and possibly the anterior ring of the first cervical vertebra are involved in a lesion that appears to be expanding the bone. There are multiple, loculated, cyst-like areas of rarefaction, with slight reaction about these cyst-like areas and with some remaining trabeculation. The skull shows no localized disease. The chest film is not remarkable except for the enlarged heart. The films of the lumbar spine and pelvis show what appears to be Paget's disease in the left innominate bone. There is an indefinite finding in one of the left lower ribs, which consists of a suggestion of faint rarefactions.

DR. ALFRED E. KRANES: What about the changes shown in the lateral views of the lumbodorsal spine?

DR. WYMAN: I think they represent old injuries.

DR. REIDY: Do you think the generalized demineralization is in excess of what you would expect it to be?

DR. WYMAN: I thought it was in keeping with that seen usually in a man of sixty-five years. I am not certain what these queer areas in the left ribs mean, however.

DR. REIDY: The physical examination gave very little help except to show the degree of spinal-cord involvement, but no idea of its cause. The laboratory findings other than the eosinophilia and the altered albumin-globulin ratio do not seem to be particularly significant. Although the patient had trauma originally, it would hardly produce the x-ray picture seen here. Occasionally, the so-called Kümmell's disease — collapse of a vertebra — will occur after a fracture, but usually not this destructive and expanding type of lesion. Trauma may have brought about further collapse of the vertebra. I think the trauma per se needs no further consideration.

Straining while coughing might have caused further trouble, but other than that I do not believe the pulmonary condition was too important. It was probably an infection since it cleared up rather rapidly in a week or so. It probably had no diagnostic significance.

As far as infection was concerned, there was some slight reaction about the changes in the vertebra, but certainly nothing in the physical or laboratory examinations to suggest an acute inflammatory process. A chronic infectious lesion, such as tuberculosis, is a possibility, but again the x-ray picture would show a different type of destruction and almost certainly a tendency toward healing in a lesion that had been present for this period. On physical examination there was no focus of tuberculosis that might have been a primary lesion.

Other possibilities suggested by the description of the demineralization are some type of hyperparathyroidism, osteomalacia and osteoporosis, but certainly there was not enough in the x-ray films and nothing in the history or blood chemical findings to substantiate a diagnosis of any of these conditions.

Metastatic disease is certainly a possibility. I have seen similar cervical-spine involvement from metastases, but there was nothing to substantiate that. The general nutrition was perfectly adequate. None of the other stigmas of a malignant lesion were present, and there was nothing to suggest it on the physical examination. No other organs seem to have been involved. I therefore believe that a

metastatic tumor is unlikely although it is a possibility.

With the description of widespread involvement one must think of multiple myeloma. Myeloma could produce this picture; frequently, it involves a cervical vertebra, often with collapse and paraplegia, and the other signs that are present here. However, the x-ray films do not show sufficient evidence of involvement generally to warrant that diagnosis. The urinary findings were not given in the protocol. If this patient had had albuminuria, Bence–Jones protein in the urine or other evidence of kidney disease, I would have more reason to consider the diagnosis of myeloma. The more-or-less decreased albumin-globulin ratio would be consistent with myeloma.

This brings me down to the eosinophilia, which is the only abnormal finding in the patient's laboratory work-up. That suggests an eosinophilic granuloma, although this disease is more likely to be present in a child or a younger person. From the description, there may have been multiple lesions, which are not inconsistent with an eosinophilic granuloma, but usually there is a localized lesion. In that same category are Hand–Schüller–Christian disease, for which the age and the findings are not characteristic, reticulocytosis and reticulum-cell sarcoma. These conditions could account for these clinical findings and also the x-ray findings. On the basis of statistics alone myeloma would be a most likely diagnosis, but with the eosinophilia as the only outstanding laboratory change I shall make my first diagnosis an eosinophilic granuloma, with involvement of the second and third cervical vertebras, and compression-fracture neuritis and myelitis.

Dr. Wyman: I wonder if Dr. Reidy would consider the possibility of giant-cell tumor.

Dr. Reidy: I think a giant-cell tumor, although much less common, is a possibility. I did not think of it in this age group. In the presence of Paget's disease, of which I was not aware until the x-ray films were presented, one might consider osteogenic sarcoma. The x-ray picture, however, certainly is not characteristic of it. There was no new-bone growth to justify this diagnosis. This was an osteolytic type of lesion.

Clinical Diagnosis

? Myeloma, cervical vertebra.

Dr. Reidy's Diagnosis

Eosinophilic granuloma of vertebra.

Anatomical Diagnosis

Plasma-cell myeloma, first, second and third cervical vertebras.

Pathological Discussion

Dr. Tracy B. Mallory: The biopsy of this lesion showed a tumor composed entirely of very well differentiated plasma cells, and a diagnosis of plasma-cell myeloma was made. This may have been a single localized lesion. If so, the prognosis is considerably better than it is for the diffuse generalized disease. A few of these localized plasma-cell myelomas have responded to surgery or even to such simple measures as curettage, with very long periods of cure.* In most cases, however, other lesions eventually appear. Operation was suggested to this patient with the intention of doing a fusion of the vertebras, but he refused and was finally sent home in a cast.

Dr. Kranes: After the diagnosis was made, was a bone-marrow biopsy done?

Dr. Mallory: A biopsy has not been done in any other spot. The urine was examined, but no Bence–Jones protein was found.

*Christopherson, W. M., and Miller, A. J. Re-evaluation of solitary plasma cell myeloma of bone. *Cancer* 3:240-252, 1950.

CASE 36432

Presentation of Case

A forty-nine-year-old man was admitted to the hospital because of abdominal pain.

On the night before admission at about eleven o'clock while on the way to work, the patient noted a mild pain across the left upper quadrant of the abdomen radiating to the left shoulder. The pain gradually but steadily increased while he was at work and was thought to be accentuated on straining. During the following day the pain grew worse. It was especially severe when he was lying down. His appetite disappeared, and he took only occasional sips of water and ginger ale during the day although he denied having nausea or vomiting. An enema taken on the morning of the day of admission had good results. There was no melena. There had been no recent increase in abdominal girth. The patient had considered himself in excellent health before the onset of the abdominal pain.

The past history was negative except for a repair of a hydrocele six years before admission. He drank four or five glasses of beer a day and about half a pint of whisky a week.

Physical examination revealed a well developed and well nourished man in moderate distress. The skin was cool and moist. The chest was clear. The abdomen was rotund and moderately tense. Deep palpation caused pain referred to the left upper quadrant below the costal margin. There was no spasm or rebound tenderness. Rectal examination was negative.

The temperature was 99.5°F., the pulse 120, and the respirations 20. The blood pressure was normal.

The urine had a specific gravity of 1.022 and gave a ++ test for albumin and the sediment contained a few red cells, a few white cells and frequent hyaline

casts. Examination of the blood revealed a hemoglobin of 10.0 gm., and a white-cell count of 11,500, with 64 per cent neutrophils. The nonprotein nitrogen was 33 mg., the total protein 6.3 gm., the amylase 27 units and the fasting blood sugar 113 mg. per 100 cc., and the chloride 101 milliequiv., and the carbon dioxide 31.4 milliequiv. per liter. The prothrombin time was 19 seconds (normal, 18 seconds). The blood Hinton test was negative. A roentgenogram of the chest was normal, and a plain film of the abdomen failed to show evidence of free air beneath the diaphragm. There appeared to be some enlargement of the spleen. The left kidney and psoas muscle were not well visualized. Only a small amount of gas was scattered throughout the large and small bowel.

In the hospital the patient continued to complain of pain below the tip of the eleventh rib on the left although he felt somewhat better. On the evening of the third hospital day the temperature was 100°F., the pulse 120, and the respirations 30. Examination at this time showed more diffuse tenderness throughout the left flank and over the left twelfth, eleventh and tenth ribs on heavy pressure. A urine specimen gave a ++ test for albumin and the sediment contained a rare hyaline cast. Examination of the blood disclosed a hemoglobin of 9.4 gm., a red-cell count of 3,250,000 and a white-cell count of 13,000, with 78 per cent neutrophils. A second serum amylase was 21 units per 100 cc. A roentgenogram of the chest showed coarse, irregular strands of increased density in the posterior basal segment of the left lower lobe. This was associated with a small amount of effusion in the left costophrenic sinus. The left leaf of the diaphragm was elevated, but its excursion was not limited. The remaining lung fields were clear. An intravenous pyelogram was normal. On the fifth hospital day a tender mass could be palpated in the left subcostal region. There was a suggestion of edema of the overlying skin in this area. A third serum amylase was 23 units per 100 cc. On the following day the patient had a spontaneous bowel movement. The mass now measured 1 inch in diameter, and was tender and easily palpable. On the seventh hospital day a plain film of the abdomen showed a marked increase in the size of the spleen in comparison to the previous roentgenogram. This enlargement caused downward displacement of the left kidney (Fig. 1).

Several hours later an operation was performed.

Differential Diagnosis

Dr. John B. McKittrick*: Dr. Wyman, could we see the x-ray films, please?

Dr. Stanley M. Wyman: The first two films, taken on admission, show a grossly clear chest and a normal heart. There is no evidence of gas beneath the diaphragm. The spleen is markedly enlarged on the first film. I cannot outline its entire extent because the film does not cover the entire abdomen. On the film taken at the time of admission, the left kidney is displaced considerably downward, and appears to overlie the left psoas muscle slightly. The film taken a few days later again shows a very large spleen and a little atelectasis in the lower-lung fields. A single film from the pyelogram shows normal-appearing upper urinary tracts but confirms the downward displacement of the left kidney. This film (Fig. 1) best demonstrates the much enlarged spleen, which seems smooth in contour.

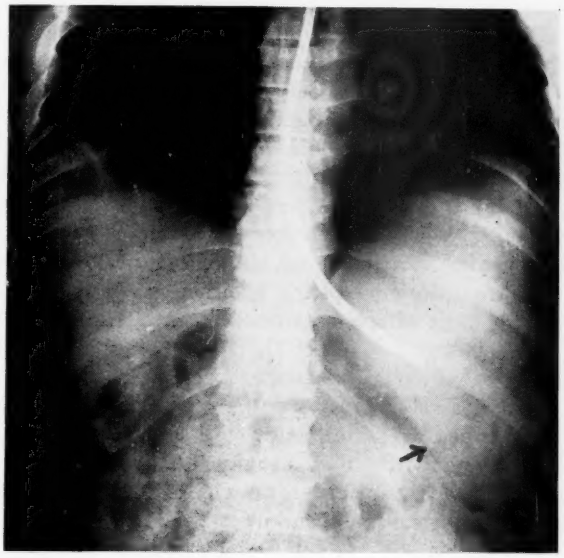

Figure 1. *Plain Film of the Abdomen.*
Arrow points to the medial margin of the enlarged spleen.

The notch can be made out. The findings in the chest as noted in the record are consistent with atelectasis, elevation of the left leaf of the diaphragm and probably a small amount of fluid in the left pleural space.

Dr. McKittrick: I am assuming that this mass in the left upper quadrant is the spleen. It certainly is perfectly consistent with the spleen in outline, and I do not know of anything that could readily be confused with it. I would like to know whether there is any displacement of the colon and if so, in what direction?

Dr. Wyman: I think, as Dr. McKittrick says, that this mass in the left upper quadrant has to be the spleen. There is gas in the colon at this point and that is probably the splenic flexure. Unfortunately, I do not have the colon filled with barium to be certain.

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DR. MCKITTRICK: Would you say that the colon is lower than usual?

DR. WYMAN: Yes; the splenic flexure is displaced downward. I cannot say whether it is displaced anteriorly or posteriorly of course.

DR. MCKITTRICK: I have to explain what I think was an acute process in a forty-nine-year-old man with no evidence of any previous illness, certainly none of any major consequence. The things I have to explain are a mass in the left upper quadrant and an irritative phenomenon under the left leaf of the diaphragm. The type of pain with radiation to the left shoulder is perfectly consistent with such a process in relation to the left leaf of the diaphragm. I also have to explain the development of anemia with a hemoglobin of 9 to 10 gm. On the basis of the chest film, I think I can eliminate disease in the thoracic cavity. A small amount of fluid in the chest and a few increased markings in the lower portions of the lung fields are perfectly consistent with a subdiaphragmatic lesion.

I should like more information to help explain anemia in a patient, previously well, who had abdominal complaints. It would be important to know whether this degree of anemia developed because of free bleeding through the gastrointestinal tract, for example, or whether it had developed on the basis of some confined internal hemorrhage. The demonstration that there was no occult blood in the stool would be most helpful.

The mass is simply mentioned at first and later is described as 1 inch below the costal margin and slightly tender. It is usually helpful to have a complete description of such masses — for example, was the mass fluctuant, was it hard, was it smooth, was it movable, and was it irregular? However, I do not have that information so that I shall have to take it for what it is worth. Also, it is interesting to me that a spleen of this size was not palpated. I have assumed that this mass might have been the tip of the spleen, but I would expect the spleen to have been down somewhat lower in the abdomen than this apparently was.

I have asked Dr. Wyman if he could tell me anything about the configuration of the colon. A barium-enema examination would have been most helpful, not necessarily to include or exclude intrinsic disease of the colon because I do not believe this was a large-bowel lesion, but to give me some idea of the relation of the colon to this mass — for example, whether the colon was pushed downward or whether the mass was anterior or posterior to the splenic flexure. That information would help me a great deal in localizing the mass and thereby guessing what its source might have been. Assuming the mass to have been enlarged spleen, I can eliminate disease in the adjacent organs such as the cardia of the stomach, the splenic flexure, the left lobe of the liver, the tail of the pancreas and the upper pole of the kidney. The pyelogram eliminates any parenchymal disease in the kidney. I do not know how to explain the casts, the red and white cells and the albumin except possibly by dehydration and low hemoglobin in an acutely ill person. To me the kidney findings do not suggest that the kidneys were the primary source of this process.

Could this have been a subdiaphragmatic abscess? I do not believe so. The history is not at all consistent with an abscess. I would expect a primary subdiaphragmatic abscess to have made the patient much sicker and to have produced more temperature. I do not believe that this was a pyogenic process. A subdiaphragmatic abscess can be metastatic from a distant source; it can be secondary to infection in some adjacent organ, but there was no evidence of infection in the history.

My opinion is, then, that this was probably a benign process. That narrows the field down but does not necessarily give me a diagnosis. The first thing one thinks of, of course, in a patient previously well is a sudden catastrophe in a benign process in the left upper quadrant such as a cyst of the spleen. Cysts of the spleen are not common, but they are not rare. They can be the site of a hemorrhage. If this was a splenic cyst with hemorrhage, it would explain the enlargement of the spleen, the anemia and the pain.

Apparently the pancreas was much in the minds of the physicians who examined this patient. The three normal serum amylase tests are strong evidence that this was not pancreatitis, but by no means do they exclude it. Pancreatic cysts usually follow some form of pathologic condition in the pancreas and are very likely to develop secondary to a pancreatitis. There was no evidence to support this diagnosis.

What about this intriguing and to me completely unfamiliar condition known as a "wandering spleen"? It is rather a romantic condition and is not very common. I do not know whether or not this patient had a spleen that was unusually mobile and that by shifting its position caused thrombosis of a splenic vein and conceivably splenomegaly. This would be a most unusual explanation for this picture.

Was this mass intra-abdominal or retroperitoneal? I think it was intra-abdominal. I need not assume that the mass was retroperitoneal just because the kidney was displaced downward because we know that an enlarged spleen will displace the kidney downward. Therefore, the only diagnosis I can make to explain the enlarged spleen, the anemia and the pain is splenic cyst with hemorrhage into it.

DR. JACOB LERMAN: There are some points that suggest infection, such as tenderness in the left flank, tenderness over the ribs anteriorly and, later, the suggestion of edema of the overlying skin.

DR. MCKITTRICK: Those things are always present with infection, but I thought that they

were not prominent enough to explain the entire picture in this case. The patient apparently was not particularly sick but just steadily and progressively became more aware of a pain. There was no evidence of fever or a high white-cell count. I cannot explain all these things on the basis of infection. Edema and tenderness certainly can be present with other phenomena that are not primarily sepsis, such as hemorrhage.

Dr. Tracy B. Mallory: Dr. Donaldson, do you want to tell us about the impression on the ward?

Dr. Gordon A. Donaldson: When this patient first entered the hospital, we thought that he had an acute pancreatitis localized to the tail of the pancreas because of the fact that he was cold, clammy and in borderline shock. He had exquisite pain in the left upper quadrant, which immediately radiated to the left shoulder whenever he tried to lie down in bed, so that he was in a good deal of agony. We watched him several days while this mass in the left upper quadrant became larger. We, too, were concerned about the anemia that prevailed. The hemoglobin fell to about 9 gm. over two to three days, and the pulse remained elevated at about 120. We did not do a barium-enema examination because we thought that this might possibly be a process originating in the splenic flexure and that we might fill an abscess cavity with barium.

Clinical Diagnosis

Perinephric or perisplenic hematoma.
? Perforated splenic-flexure diverticulum.

Dr. McKittrick's Diagnosis

Splenic cyst, with hemorrhage into cyst.

Anatomical Diagnosis

Rupture of spleen, probably through splenic cyst.

Pathological Discussion

Dr. Donaldson: The operative incision was made over the splenic area, and on opening the peritoneal cavity, there was a considerable amount — approximately 1000 cc. — of clotted blood present. The spleen was visualized and found to be enlarged; in addition there was a membrane that was very adherent to the undersurface of the diaphragm and the lateral abdominal wall. The membrane seemed in places to be continuous with the spleen as though it might have been a portion of the capsule. A good deal of time was spent in retrieving this membrane from the diaphragm and the lateral abdominal wall. The spleen was removed with the membrane.

Dr. Mallory: The spleen as received in the laboratory was slightly but not markedly enlarged, weighing about 400 gm., and showed a very long rent in the capsule with a significant amount of subcapsular hemorrhage. It appeared otherwise normal. Microscopically, there were none of the pathologic processes that are commonly associated with a spontaneous rupture of the spleen, such as leukemia, infectious mononucleosis and once in a while, hepatitis, and the exact reason for the splenic rupture was not obvious from the specimen. We were inclined to think, as Dr. Donaldson did, I believe, at the time of the operation, that there must have been a cyst in the region of the upper pole into which the hemorrhage had occurred and then secondary rupture of the cyst wall. We were, however, unable to demonstrate an actual cyst wall to our own satisfaction.

Dr. McKittrick: I would think that it would be more common than just spontaneous rupture of a perfectly normal spleen; would you not, Dr. Mallory?

Dr. Mallory: The latter is extremely rare, of course. In patients with enormously enlarged spleens a rupture does occur apparently spontaneously or with minimal trauma, but this spleen was not of the dimensions that would make that reasonable. I think the most likely cause of an apparently spontaneous rupture of a spleen is infectious mononucleosis, which I believe we can exclude here.

NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-NINTH ANNIVERSARY

House of Delegates, September 10, 1950

THE House of Delegates convened at the Mount Washington Hotel, Bretton Woods, on September 10, 1950, at 2:00 p.m., with Dr. B. Read Lewin, of Claremont, as speaker.

The following delegates answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Roger P. Brassard, Laconia
W. J. Paul Dye, Wolfeboro
Ralph W. Tuttle, Wolfeboro (alternate for Francis J. C. Dube)
Albert C. Johnston, Keene
Louvane A. Fox, Keene
Julius Israel, Berlin
Marjorie Parsons, Colebrook
Everett C. Campbell, Woodsville
Reginald K. House, Hanover
Leslie K. Sycamore, Hanover
Leslie E. McKinlay, North Haverhill
Harris E. Powers, Manchester
Richard T. Mulvanity, Nashua
Raymond H. Marcotte, Nasaua
Frank F. Flagg, Nashua
Edward D. Hagerty, Manchester
Lloyd L. Wells, Manchester, (alternate for Oscar Burns, Milford)
Warren H. Butterfield, Concord
Edward V. Putnam, Warner
William D. Penhale, Concord
Francis Brown, Henniker
Frederick S. Gray, Portsmouth
Louis C. Theobold, Exeter
Henry Saltonstall, Exeter
Albert E. Barcomb, Rochester
Addison Roe, Newport
Pauline Spear, Claremont (alternate for B. Read Lewin, Claremont)

The speaker declared a quorum present. The President appointed Ralph W. Tuttle as alternate for Francis J. C. Dube, Lloyd L. Wells of Manchester as alternate for Oscar Burns, and Pauline Spear of Claremont as alternate for Speaker Lewin.

On motion duly made and seconded, it was voted to omit the reading of the minutes of the previous meeting, because of the publication of the proceedings.

The speaker made the following appointments: to the Committee on Credentials, Drs. Israel, Fox and Putnam; to the Committee on Officers' Reports, Drs. Brown, Walck and Roe; to the Committee on Communications and Memorials, Drs. Gifford, Hayward and McCooey; and to the Committee on Nominations, Drs. Gray, Dye, Powers, Butterfield and House.

The President then presented the following report to the House of Delegates:

> In the development of our present social order there is no profession, business, or trade that is not organized.

Factually, the medical profession was one of the first. Its purpose was and is the development and the protection of the proper medical care of the American people.

The emphasis heretofore has been on the "development" — by the stimulating and sponsoring of advances in medical science, and then by disseminating their application by various educational technics. Our accomplishment in this phase is a matter of record. Now we are confronted with the urgent necessity of "protecting" that medical care — against the onslaughts of socio-economic enthusiasts whose tendency to misguidance is brought out by their basic, fallacious concept that medical care, with all that contributes to it, can be regarded as a commodity. The purpose of organized medicine was not and is not the selfish protection of us as individuals. Herein lies the entire basis of our problem of public relations.

We find ourselves confronted by organized opposition. Organized effort on our part is therefore unavoidable, and if we are to recognize our obligations, it is inevitably necessary. The medical profession must come out of the seclusion of its internal operations and enter the public arena, whether it likes it or not. Medical men are recognized as men of science, but our standing as men of service must be maintained.

What underlies the present issue is quite simple and clear. Medical advance in itself has created higher costs to the point that the cost of modern medical care has become an economic problem in the minds of so many of our people that it has become an issue of public policy. By that step, it has moved into the status of a political issue. This issue has centered on the application of the insurance principle to the American family's problem of medical-care expense. Clearly, the issue at this point is whether this process is to be governmentally controlled or to be developed on a voluntary basis. The first leads to national bureaucratic control; the second can preserve those principles in which we believe. Any attempt to confuse this final issue by our arguing such questions as the American family's expenditures for unnecessary items is beside the point, impractical, and ineffective. Only as points in an educational campaign for proper orientation of the problem are they important.

The activities of the Committee on Public Relations are reported by that committee. Initiated by a previous house of delegates, this program has been effective. But we can neither survive nor fulfill our obligations by merely defeating other people's bills. There must be some constructive contribution. The American Medical Association has taken its position definitely on the side of voluntary insurance.

In fighting the national health program, we are battling the socialization of medicine — first, as poor medicine, and second, because historically this development has indisputably led to the complete socialization of a people. This program demands organized activity in two aspects as applying to our society. One is external, and the other is internal. On the external we have made progress. On the internal in the sense of development of a program of constructive, positive contribution and leadership, we have only begun.

Theodore Roosevelt once said it was the obligation of every member of an organized profession, business, or trade to contribute some portion of his time to the efforts of his organization, assumed to be working for the common good. We have plenty of such men. But we have landed in this present phase with the most archaic constitution and by-laws now existing among medical societies. Specifically, for instance, our Society has no provision for preparing officers for their executive duties or the machinery for the proper implementation of the job. There has been but one continuing officer. Over the

past thirty-five years, the Society has had but two secretaries, who have had to assume the entire propulsion of the business of the Society. Let it be said at this point that they had done it well and they had fulfilled to the benefit of the Society an oftentimes thankless duty. With the resignation of Dr. Metcalf, we imposed on Dr. Deering Smith, already elected Vice-President and American Medical Association delegate, to take over the Secretaryship pro tempore as the person at the time most familiar with the affairs of the Society. An executive committee was appointed at that time consisting of the officers and the first past-president. The appointment of an executive secretary was made after approval by the House of Delegates.

We believe that a basic need of the Society is the interest of its individual members. This can be stimulated only by wider participation in its activities and administration. We have accepted the responsibility of making general recommendations to the Committee on Revision of the Constitution and By-Laws. There is need of a President-Elect with definite succession including the Vice-President in order to provide indoctrination by a reasonable period of preliminary membership on the Executive Committee, which should have permanent status. It should serve as advisory to the President, as the executive group of the Society between meetings of the House of Delegates, and should prepare the annual budget for submission to the House of Delegates. Because the activities of the Committee on Public Relations are involved in both basic policy interpretation as well as budget considerations, its functions might well be transferred to the Executive Committee.

The detail business of even a small medical society, such as ours, is ever increasing. The need of an executive secretary is apparent. By far the great preponderance of detail can be cared for by this officer. As a paid employee of the Society under the direction of the Secretary of the Society and the Executive Committee, the position is in its proper relation to the officers and the Society. This allows proper control and direction, and avoids the unnecessary and unreasonable demands on a practicing physician. The offices of secretary and treasurer should be separated, both lessening the duties of individual officers, to allow for further participation by more individuals, and to lessen the total cost to the Society. There is nothing radical or original in these proposals. We find ourselves almost the only society operating otherwise.

There is need for a complete review of the setup of the various committees appointed by the House of Delegates. This could well be referred to the next executive committee for study and subsequent report to the House of Delegates. Because of contingencies this year, several interim committees have been appointed. Boundaries of performance should be established to avoid overlapping or duplication. At the same time, sufficient committees should exist to avoid heavy committee loads and to provide wide participation by the membership. The method of appointment of the Nominating Committee has been criticized. It could well be a representative elected by each County Society with the President-elect as chairman, who could logically be given the privilege of participating in the formation of the administrative setup with which he is to operate. In this connection, we should like to record the observation that greater participation in the determination of state-society administration be given to county-society action. For instance, the prestige and effectiveness of the Council would be rejuvenated to a considerable extent were the councilor, as representing a county society, elected by that county and not by the House of Delegates.

Such a procedure was followed in the establishment of an interim Blue Shield State Committee. Its need was early encountered — to bring the Society membership into immediate contact with and participation in the policy-determining procedures of Blue Shield. The Blue Shield Grievance Committee program was adopted by the American Medical Association last winter. A committee appointed by the Speaker of the House is to report on such a program at this annual meeting.

The combined meeting this year with the Vermont State Medical Society, as approved by the House of Delegates, should provide a desirable trial attempt at a more ample size of meeting. It should be more satisfying to exhibitors and speakers, and will provide an opportunity to discuss with our neighbors problems that are common to both groups.

Budget-wise, we are proceeding within bounds. The collection of dues still leaves something to be desired. Further explanation of their origin and need should be unnecessary repetition. There seems to be a tendency on the part of some to regard dues as a charitable contribution rather than the obligation of a member of a professional organization. On the basis of the present year's experience, it is possible that the program might be carried out at this point with a reduction in the dues. It is our opinion, however, that it would be best continued on present rates with the probability of accumulating increased reserves in this immediate period.

At the beginning of our term, it seemed that the important project ahead was the effecting of Society organization to meet more adequately the changing demands on the Society in the present state of medical-care problems. However, several unanticipated conditions and incidents arose that consumed considerable time and energy. We should like to recommend that to some group, perhaps the councilors, be referred a study of the determinant of the sub-unit of the State Society, now the county societies as determined by county lines.

Geographic county lines have little to do with normal flow of medical care or community of interests of the membership within those lines. The result is too wide a range in size of membership, occasional relative isolation of small groups, and in some instances too large an area for reasonable travel to county meetings. This situation could well be surveyed.

Dr. Brown, for the Committee on Officers' Reports, moved that the complete review of the setup of the various committees, as recommended by the President in his report, together with his suggestion to have the Nominating Committee elected by the county societies, be referred to the next Executive Committee.

This motion was duly seconded and, after some discussion, carried unanimously.

The President then made the following remarks:

I should like to make one brief report on the military situation that has developed in the past three weeks. That is not only to protect myself and the Executive Committee but also so that everyone will understand what there is to be known, which is surprisingly little.

As stated in the survey letter that went out on the eighteenth, the presidents of the Societies of the First Army Command were called to Governor's Island, on a moment's notice, really, and we sat there for six hours, and they discussed their problems and their needs and lack of medical personnel, and the urgency of the situation. It was rather confusing.

We were asked at that time to get volunteers from the ASTP in each of the states. We all know what that has been for the last year. The American Medical Association tried to do it; the Army tried to do it, and out of thousands, they got 3. That has not pleased the Army personnel, of course, so we were asked what we could do about it. Nobody thought anything could be done, but all offered to write letters, stating the case, and then see what would happen.

We had, at that time, in New Hampshire 6 men in that category; we got 1 volunteer, which is a high percentage, as compared to the experience in the past. Then, we were actually not asked, but it was implied that each president should go back to his executive group and make recommendations about reserve officers.

That was on Thursday. We were given a week to send these in.

Rhode Island got going rather early, and, we understand, sent in a list of their reserve officers, and with how many recommendations we do not know, but these men were immediately notified that they were recommended

by the Society, whereupon there was a small-sized riot in Rhode Island.

Massachusetts, immediately when they got into action, sent a telegram to the Army and to many other agencies related in this problem, and they refused to do anything.

We were called up and asked where our list was. We said that we were sorry, but there was not any list. As a matter of fact, they gave us a list of their reserve officers by states; we have 15 in the State that are technically reserve officers. They are largely the present war reserve officers, or men who went into the reserves as volunteers at the time of the mobilization. It was quite evident that the list was not up-to-date, because I saw 2 men listed as captains who, I happened to know, had been discharged as lieutenant colonels, which began to make one a bit doubtful of the current accuracy of the records.

We then sent a wire, of which I have a copy, in order to be certain that we were on record as to what we were doing; this was sent on August 29, which is the day that I was called up and told that we had to have the list in their hands.

Medical Section
Headquarters, First Army
Governor's Island
New York 4, N. Y.

Executive Committee New Hampshire Medical Society unwilling to assume arbitrary and nonexistent power to recommend four men out of total of nine eligible according to list submitted by Headquarters office. Consider time allowed totally inadequate to permit proper survey and proper briefing of membership.

JOHN P. BOWLER, *President*

That refers back to the fact that we had a quota of 4 general medical officers in their scheme of things, and they told us they would be taken at or below the rank of captain. We did not send in a list, or any recommendations.

In spite of all that, I think I have been telephoned and have seen and have been called upon by most of these reserve officers, who were called up for their physical examinations in the past week and in two or three instances, they were shown a paragraph in Army orders in the Adjutant's Office in Manchester, where they had been recommended by the New Hampshire Medical Society. I think it did say the "Medical Society"; it was apparently a form that they could use as a universal proposition.

Last Friday night, just before leaving home, I received the latest copy of the bulletin of the American Medical Association on the military situation at Washington, and I have copies of the latest information with me. This is the latest and the most accurate review of the military draft bill, which went through last week.

I should like to have you read, also, the letter that I wrote myself to each of these men whose names are given us by the American Medical Association, because all these men got all their training under the Government in one way or another, and have not served, and they are in the files of the American Medical Association. We have 6 of those, so far as we know. There may have been some changes, of course, and some of these men might have moved, so that all the data probably are not accurate. But I am doing this in order to leave no question mark about what has gone on, and the reasons for it.

First off, I would be most anxious that you did not misinterpret this letter. In the present status of things, neither the New Hampshire Medical Society nor myself as President has any authority or any power, and, as a matter of fact, is unwilling to assume arbitrary power in the process of selection of medical men for military service until such time as a more over-all plan has been worked out. This is merely a matter of relaying information derived from contact with the military authorities, and is to be used as you see fit.

We have no private information, and all that we were told in New York has been stated publicly and in the Journal meanwhile. Through the Emergency Medical Committee of the American Medical Association, we are kept in daily contact with the developments in the man power control situation.

The probability is nearly a certainty that the younger medical men trained under the government program and who have had little or no military service will be called up for physical examination and, if passed, for activation. Such a bill is reported as having been passed by the Senate yesterday, and is expected to be an early matter of legislation. Without discussing the merits of the situation, this legislation has the support of the American Medical Association and the urging, certainly of those Medical Reserve Officers who have already served considerable military service in the past and who are now being reactivated.

For those men who volunteer before such an action goes into effect, there is the probability of a more desirable allocation and the certainty of the $100 a month extra pay, which is not effective to those men who are called.

In any event, we are asked to give out the warning that no man who wishes to volunteer make any radical provisions in his planning until he will have been sent for and passed a physical examination, after which he will be given sufficient time in which to execute his own plans. If you are interested in volunteering at this time, will you get in touch with me, or directly with the Medical Section, Headquarters, First Army, Governor's Island, New York 4, N. Y.?

I would merely repeat the thought of my first paragraph, that if I can be of any help in the way of information, let me know.

JOHN P. BOWLER, M.D.

P.S. In the course of writing this letter, this bill has now been passed by the Senate and House, as you probably read yesterday. J. P. B.

There has been no great happiness about the calling of these men, even for physical examinations. I have seen the orders, as perhaps most of you have; they stipulate the men will be activated for physical examinations and then out of those will be selected a quota.

Now, this is to be made clear from the technical point of view. A reserve officer is not in this draft registration or anything else. He is a reserve officer, and the only agency that can do anything about that, so far as I know up to the moment, is the Army.

So after the second man came back from Manchester or was on his way to see me and ask me what I knew about this, I was told in each instance that they had been shown a paragraph in some order or form whereby they had been called up on the recommendation of the New Hampshire Medical Society. Then, in the presence of one of these men, I telephoned the Executive Officer in the Medical Section of the First Army and asked him if he received our wire. He said he did.

You can see where I was perfectly willing to take the position that there was a lot of buck-passing going on. I said to him: "You have a copy and I have a copy. Is that right?" He said it was. I said to him: "I am sitting right in front of a reserve officer who has come to see me, and he has been presented with a letter at the Manchester office that said that he was recommended by the State Medical Society." Then he said: "Well, I don't understand that. We told you fellows we were hoping for recommendations." I said: "Yes, and we certainly answered you that it was not within our power to call up a reserve officer, and the only person we knew who could do it was the president of the United States or his legal agent."

Whereupon he said that he would look into it and then he did, I must say, have the graciousness to write the following letter:

Reference our telephone conversation yesterday, I immediately called the New Hampshire Military District in Manchester, with reference to the letter that you mentioned.

At the time we had our initial conference at Governor's Island with the presidents of the State Medical Societies, a letter was sent to all of the State Military Districts informing them that the State Medical Societies had been requested to assist us in making the selection of Reserve Officers for recall at this time. This is the letter that the office had in their possession. It did not state that the New Hampshire State Medical Society had assisted in the selection, but that it had been requested to do so. I asked them not to show the letter

to any others, in view of the misunderstanding that had ensued.

We regret that you have been embarrassed through this misunderstanding.

I think it is very important that every one know the facts. And at least this phase of it has been ended. The present bill may be summarized as follows:

Physicians and dentists *who have not reached the age of fifty* and *who are not members of military reserves* must register — military reserves are already subject to military orders.

Any registrant is subject to induction if acceptable to the military. The law provides that registrants will be called up on the following priority:

1. Former ASTP and V-12 men *who have not served on active duty* (military, Coast Guard or Public Health Service) and others *deferred from service to continue their education* during World War II, and who have had less than ninety days of active duty (military, Coast Guard or Public Health Service).
2. Members in the above groups who have had more than ninety days' active duty (military, Coast Guard, or Public Health Service) but *less than twenty-one months.*
3. With no reference to above groups, those who did not have active service (military, Coast Guard or Public Health Service) subsequent to September 16, 1940; this could include postwar medical graduates as well as other physicians who have not served and have not reached their fifty-first birthdays.
4. All others, including World War II nonreserve veterans, men in this group to be called on basis of extent of duty (those with least duty first and so forth).

A registrant can get a Reserve Commission if he is acceptable to military. If he applies for and is granted a reserve commission, he will come under military orders, and Selective Service will not process him further.

Every reserve officer called to duty, on voluntary or involuntary basis, receives the extra $100 per month. Men required to register under this act also may qualify for the $100 — but only if they volunteer prior to their actual induction.

Actual deferment is at the discretion of local Selective Service boards. However, the law states that "the President is authorized to provide for" certain deferments. Action under this clause would be based on the registrant's previous military service, his dependency status and any undue hardship that might ensue.

The law also provides for establishment of a national advisory committee "which shall advise SS system and co-ordinate work of state and local volunteer advisory committees with respect to selection of needed . . ." professional personnel. The committee will be composed of men "outstanding in medical, dental, and allied services." *Medical and dental professions must be represented on the national committee,* but representation of other professions is not required. The law does not control local advisory committees; whether these are established and made effective depends on local conditions.

The law itself attempts to set up safeguards. It says that the national and state and local committees shall "*give appropriate consideration to" civilian as well as military, medical and dental requirements.* Another section underscores this policy: "Maintenance of national health, safety or interest" should be considered in granting deferments. In calling up reserves, *Army commanders* are also under orders to give careful consideration to doctor-shortage areas.

Military medical officers hope enough men now will volunteer to meet requirements. They are prepared to use the law's authority for mandatory inductions, but are confident that these cases will be rare. They are hopeful that the $100 bonus, which is denied to men involuntarily inducted under this act, will attract registrants to volunteer.

One section of the law authorizes transfer of medical officers from one service to another, with consent of the officer and of services involved. Officers are protected in promotion, retirement and pay. Another provides that any person who has served in the armed forces or Public Health Service after September 16, 1940, and is recalled, may be "promoted to a grade commensurate with his education, experience and ability." The law states "it is the sense of Congress" that Selective Service deferments will be granted to premedical, predental and allied students in numbers equal to the present such enrollment. This ruling more or less formalizes present Selective Service policy and sets a total figure for deferments in each professional category. Inductees under doctor draft are required to serve *twenty-one months.* Anticipation is that the Advisory Committees will operate like Procurement and Assignment of World War II.

Dr. Brown then stated that the Committee on Officers' Reports recommended a study of the redistricting of the Society, but that the Committee did not recommend that any action be taken, since it considered that the problem was adequately covered for the present by the *By-laws*, Chapter 14, Section 8.

The Committee had decided that when any specific action was recommended, each recommendation be acted upon. And, if a committee report was adopted, all recommendations contained therein would be adopted. Dr. Brown moved that the President's report to the House of Delegates, be recorded in the minutes of the meeting.

This motion was duly seconded and was carried.

Dr. Brown then asked whether it would be advisable for the officers, the President or the Executive Committee, or some other committee, to suggest the names of members of the Society to the military authorities, when asked for in a manner similar to the procedure under the Procurement and Assignment setup in the war. The President either should or should not have the authority, and he should know what he might do in the matter. The medical profession had to co-operate with the military authorities to a certain extent, and the military authorities should know where to go, or else the Society would not have any say in the matter.

Secretary Smith stated that in World War II President Jones was asked to suggest to the Secretary and the President of the American Medical Association someone to serve on the National Emergency setup. He suggested Dr. Smith, who was appointed as the New Hampshire representative on the Committee. Later, when that committee was taken over by the federal Government, Dr. Smith was made chairman of Procurement and Assignment in New Hampshire, and asked to appoint two additional members; later this committee was enlarged to five members, who served throughout the war, to get men into service and also into areas where they were needed in a civilian capacity. Serving with him were three to five doctors from each county.

In reply to a question Dr. Brown stated that the only committee with similar authority at present was the Committee on Emergency Medical Service, but there appeared to be no statement of authoriza-

tion in the *By-Laws* or in the Constitution of the Society giving the committee any authority.

Secretary Smith said that the Committees on Emergency Medical Service had been set up in each county, for civil defense only. Nothing further had been said, in any information that he had received from the National Emergency Medical Service Committee of the American Medical Association. He had been asked to get each county society to appoint a civil defense committee. That had been done in every county. The Committees were working under the state committee of Dr. Sullivan.

Another question concerned the law to be signed by the President providing that a national advisory committee shall be established "which shall advise Selective Service system and co-ordinate the work of state and local volunteer advisory committees with respect to the selection of needed . . ." professional personnel.

President Bowler stated that the proposed bill applied to registration. The Lawrence office in Washington was already in the process of developing a plan of medical participation in the screening. He believed that the only thing to do would be to continue the position already taken and then wait for developments of the governmental machinery, which, he believed, was being set up. Undoubtedly, the American Medical Association was insisting upon working out some participation along the lines of that in the last war.

The Executive Committee had been asked to submit three recommendations of names, from whom they would pick one as chairman.

Dr. Saltonstall asked if, in the last war, the armed forces were given the names of people who were regarded as available.

Secretary Smith replied that that was part of the function. The rest of it was to tell the armed forces those who were not available. Dr. Smith had told them more men were not available than were available.

Dr. Dye stated that under Chapter VI, Section 1, of the *By-Laws* the President was directed to appoint all committees not otherwise provided for. Until such time as the national setup was ready he moved that this question be tabled, and that when such a committee was appointed, men with past service be on it.

The motion was duly seconded and, on a standing vote of 16 to 10, was carried.

Dr. Brown then moved that the position of the president of the Society during the past month be approved.

This motion was duly seconded and was carried.

Dr. Smith then presented the report of the Secretary-Treasurer, as follows:

Your secretary wishes first to record the great loss to the Society in the death of two of our officers: Howard N. Kingsford, trustee and past-president; and Donald W. Leonard, vice-speaker of the House. Both have been very active in organized medicine, and they will be missed at this annual session.

The year has been a busy one for the Society, with three special sessions of the House of Delegates and a vigorous educational and public-relations campaign. There have also been numerous meetings of the executive committee, created and appointed by President Bowler.

I have attended at least one meeting of the majority of the county societies; the meetings of the Council of the New England State Medical Societies in Boston; the Conference of State Medical Association Secretaries and Editors and the Public Relations Conference — both held in Chicago; and the sessions of the American Medical Association in Washington and San Francisco. Numerous conferences were held with the Executive Secretary, committee chairmen, county secretaries and the editors of the *New England Journal of Medicine*. I have also represented the Society at several formal functions.

Two meetings of the Committee on Jurisprudence have been held to consider threats or claims of malpractice. Every member should notify our counsel, Mr. Sulloway, or the Secretary, as soon as any claim is threatened. Only one suit against a physician was tried in court, and that ended with a decision for the defendant.

Several meetings of the program committees of the New Hampshire and Vermont State medical societies have been held, and the program of this meeting is the report of the Committee on Scientific Work.

The Committee on Publications has not met. The House voted not to publish the papers read at the meetings of the Society, and the only duty left was the reading of the proof of the report of the annual meeting. Because of the time element, this has been done by the secretary. If the executive secretary continues to edit *The Bulletin*, and this seems advisable, I can see no need for continuing the committee.

Since the Treasurer's report was made at a special session of the House of Delegates, accepted, and printed in the *New England Journal of Medicine*, it will not be given here. The books of the Treasurer have been audited and found correct by a certified public accountant.

Dr. Bowler and I were guests and speakers, last fall, at the annual meeting of the New Hampshire Pharmaceutical Association. A special committee from this association has been appointed to meet with the Allied Professional Relations Committee in an endeavor to improve the present good relations that exist between the two professions.

The New Hampshire Dental Society gave our Society $600 to aid in the educational campaign.

In addition to the new committees on Alcoholism, Allied Professional Relationships, Blood Banks and Diabetes and Its Detection, all of which have been reported, Dr. Bowler appointed a committee on arthritis and rheumatism with the following members: Sven M. Gundersen, Hanover; Clarence E. Dunbar, Manchester; J. Dunbar Shields, Concord; Philip B. Daniels, Keene; and James Heyl, Exeter. The trend in our Society is toward the creation of a special committee for every chronic disease, with a corresponding committee in each of the county societies. I believe that we should consider having a committee on chronic illness, which could appoint sub-committees as it deemed necessary to study various diseases.

To stimulate the interest of the members of the several standing committees and to increase the value of their work, I suggest that a member of each committee be sent to its respective national conference.

In accordance with the vote of the House, $1000 has been given to the Trustees for the Benevolence Fund. The following woman's auxiliaries generously contributed to the fund: Hillsborough, $25; Merrimack, $25; and Strafford, $10.

Delegates were sent to the annual meetings of each of the other New England state societies, as requested by the House of Delegates. If the custom is to be continued, I wish that members interested in representing our Society would inform me, and they will be designated delegates.

The Conference of Presidents and other Officers of State Medical Societies and the Council of the New England State Medical Societies are excellent and should be sup-

ported by the Society with an appropriation of $10 for the former and $100 for the latter.

Your secretary has been appointed a member of the Committee on Legislation of the American Medical Association, a member of the editorial board of the *New England Journal of Medicine* and a consulting member of a committee of the New Hampshire Medico-Legal Society to study the medical referee system in this state. Fortunately, these appointments have not required the expenditure of much time, for the routine work of secretary-treasurer, such as correspondence, reports and questionnaires, has been steadily increasing. In addition to this, I have brought the mailing list up-to-date, set up a new file of new dues record cards for each member, kept a double-entry system of bookkeeping and sent out membership cards as the dues have been received by me. The most arduous of these has been the collection of dues. The following table shows our total membership and the results by counties, as of August 1, 1950:

County	Membership			Unpaid Dues and Assessments			
	total	active	life	'49	'49A	'50	A.M.A.
Belknap	41	38	3	1	11	14	15
Carroll	18	17	1	0	5	7	8
Cheshire	38	37	1	1	6	7	28
Coos	37	33	4	5	10	10	26
Grafton	110	106*	4	2	14	20	17
Hillsborough	172	166†	6	3	24	35	107
Merrimack	80	78	2	3	10	28	58
Rockingham	68	65‡	3	0	3	7	41
Strafford	50	47	3	0	0	0	27
Sullivan	23	21	2	0	2	2	1
No county	3	2	1	0	2	2	2
Honorary	3	0	0	0	0	0	0
Totals	643	610	30	15	87	132	330

*16 Resident — dues $5.
†1 Resident — dues $5.
‡1 In service — dues remitted.

The 1949 membership is the largest in our history but this year there will be a number of resignations and many more life members because of the increase in the dues. Failure to pay the membership dues of the American Medical Association has no effect on membership in our Society, but the total amount to be paid for county and state membership plus the American Medical Association membership and fellowship — at least $78 — has staggered some members. In order to keep the Society solvent, we must pay our dues. Some county secretaries are slow collectors, and the possibility of having this society collect its dues should be considered. The county societies have already proposed twenty men for life membership and if every physician who is eligible for life membership is granted it, the books will not balance. I believe that men in active practice should continue to pay dues and that the *By-Laws* should be amended so as to give life membership only to those who meet the present requirement of sixty-five years of age and fifteen-year membership and who are also retired or for whom the payment of dues would be a hardship.

The educational campaign of the American Medical Association is to be continued in the coming year — but with a greatly decreased tempo. The Department of Public Relations is to be expanded so as to take over this work eventually. It may be possible to make our Committee on Public Relations a real working committee and to follow the lead of the American Medical Association in handling this work.

At the last annual session it was voted that the Committee on Amendments to the Constitution and By-Laws be instructed to prepare an up-to-date copy of the *Constitution and By-Laws* to be submitted to the next House of Delegates and after review by the House that the Secretary be authorized to incorporate them in a printed booklet for the reference of the members. At the request of Dr. W. J. Paul Dye, chairman of this committee, I have read the *Transactions* from 1935 to 1949, noted the changes that have been made in the *Constitution and By-Laws*, and forwarded them to him. I have also taken the liberty of suggesting to him a number of changes in addition to those given in this report. For example, there should be provision for a president-elect and an executive committee, while the matter of committees should be clarified and brought up-to-date. Some of these changes can be made at this session, but others will require more study. I believe that our whole *Constitution and By-Laws* should be studied, changes proposed, our legal adviser consulted, and copies of the new draft sent to the delegates well in advance of the next session, at which time action can be taken on the *By-Laws*.

In performing the duties of secretary-treasurer I have had the help and co-operation of our president, trustees, executive committee, executive secretary and public-relations counsel, legal counsel, county secretaries, committee members and many others, to all of whom I wish to express my appreciation.

Dr. Smith then proposed the following change in the *By-Laws*, Chapter 1, Section 5:

> Any physician who has been a member of the Society fifteen years, and who is, through disability, unable to engage in the active practice of medicine, or who has retired from the practice of medicine, on the request of his county society, may be made a life member by vote of the House of Delegates, this membership to cease if he should resume the active practice of medicine.

The Secretary had a second suggestion for another class of membership, to include interns and residents and also physicians who were full-time teachers and who were not practicing medicine, like several at Dartmouth Medical School, but who were required to pay the $40 dues. The House had voted to make the dues of the interns and residents $5 a year. A similar suggestion was made that the teaching physicians pay only nominal dues — about $5 a year. The Secretary moved that these two suggestions be referred to the Committee on Constitution and By-Laws.

President Bowler raised the question whether these categories would have voting privileges. Personally, he thought they should not have voting privileges.

Secretary Smith accepted the suggestion in his motion, moving that the recommendations be referred to the Committee on Amendments to the Constitution and By-Laws, the first one relating to life members, and the second to the possible establishment of associate membership, to include first, interns and residents, and secondly, full-time teachers who do not practice, and that associate members do not have the privilege of voting or holding office.

This motion was duly seconded, and was carried, with two dissenting votes.

Dr. Brown moved to continue sending representatives to other New England State Society meetings.

This motion was duly seconded and was carried.

Dr. Brown then moved that the report of the Secretary-Treasurer be recorded in the minutes of the meeting.

This motion was duly seconded and was carried.

Dr. Tuttle then presented the report of the Necrologist, as follows:

The following members of the New Hampshire State Medical Society died in 1949:

Name	Address	Date of Death
Dr. Edson M. Abbott	Rochester, N. H.	May 14, 1949
Dr. Charles Adams	Franklin, N. H.	March 12, 1949
Dr. Benjamin W. Baker	Laconia, N. H.	August 17, 1949
Dr. John J. Brosnahan	Keene, N. H.	October 29, 1949
Dr. Damase Caron	Manchester, N. H.	June 9, 1949
Dr. Albert S. Dolloff	New Hampton, N. H.	March 6, 1949
Dr. Malcolm A. H. Hart	Milton, N. H.	January 24, 1949
Dr. Francis B. King	Derry, N. H.	July 11, 1949
Dr. Harry M. Morse	Peterborough, N. H.	October 27, 1949
Dr. Frank Byron Perkins	Derry, N. H.	June 21, 1949
Dr. Annina C. Rondinella	Wellesley, Mass.	March 11, 1949
Dr. Karl L. Smith	Laconia, N. H.	November 6, 1949
Dr. Frederick C. Sweeney	E. Jaffrey, N. H.	June 22, 1949
Dr. George Albert Weaver	Bradford, Vt.	June 22, 1949

The following members of the New Hampshire Medical Society have died since January 1, 1950:

Name	Address	Date of Death
Dr. Simeon Louis Hebert	Manchester, N. H.	January 17, 1950
Dr. Hiram L. Johnson	Franconia, N. H.	June 12, 1950
Dr. Howard N. Kingsford	Hanover, N. H.	February 15, 1950
Dr. Joseph E. Larochelle	Manchester, N. H.	April 3, 1950
Dr. Zenon A. Lavoie	Manchester, N. H.	June 1, 1950
Dr. Donald W. Leonard	Exeter, N. H.	February 21, 1950
Dr. Edward R. B. McGee	Berlin, N. H.	May 24, 1950

R. W. Tuttle

Dr. Brown, for the Committee on Officers' Reports, moved that the report of the Necrologist be accepted.

The motion was duly seconded and was carried.

Dr. Smith then presented the report of the delegate to the American Medical Association, as follows:

During the past year there have been two sessions of the American Medical Association. The clinical session held in Washington, D. C., December 6-9, 1949, was very well attended, and the papers and exhibits were excellent. It is hoped that there may be a clinical session in Boston in the near future, so that more of us will be able to go to it. Your delegate served on the Reference Committee on Executive Session.

The Committee on Blood Banks, of which I am a member, reported that the study of blood banks, over 1600 in the United States, is progressing well. It was stated that in a Red Cross blood center, the local control must be by the county medical society. The medical profession must participate to the fullest extent in the development of such a center and participate actively in its affairs in order that medical control may be maintained. A co-ordinated program on the national level, with complete integration of private and hospital blood banks with the Regional Red Cross blood centers on the local level and an informed and trained medical profession, will be able to meet any emergency that may arise.

The first report on the blood banks is complete now; but in view of the present international situation, a second brief questionnaire is being sent to county secretaries and blood banks. Please see that these questionnaires are filled out, for their early completion will aid in planning for an emergency. We have the full co-operation of the American National Red Cross and the Office of Civilian Defense, while Dr. Holyoke's special committee on blood banks is studying the local situation and will make a report at this session.

Membership dues of $25 were voted for 1950, and these are to be entirely separate from the Fellowship dues of $12 which include the *Journal* or some other publication of the American Medical Association. At the San Francisco session dues of $25 were voted for 1951, and *these are to include a subscription to the Journal.* The Fellowship classification may be continued; if so, the dues will probably be $2 a year. In the discussion relative to the remission of dues it was decided that in general the dues would be remitted on the same basis that the dues of county and state societies are remitted, but that physicians in active practice must pay dues.

The report of the Committee on Hospitals and the Practice of Medicine, adopted at Atlantic City last year, presented legal hazards and was referred back to the committee. The second report was discussed at San Francisco in June and adopted as amended. This report, which denounces systems whereby hospitals hire salaried physicians for medical care and bill the patients for this care, is printed on pages 1090-1 of the *Journal of the American Medical Association*, July 22, 1950. Early this year our Society appointed a committee on hospitals and professional relations as recommended in the report.

The Council on National Emergency Medical Service made a report, but I shall leave all discussion of this subject to our corresponding state committee.

The name of *Hygeia* has been changed to *Today's Health* and attempts are being made to continue the improvement of the magazine.

The following timely resolution was adopted:

Whereas, Several constituent state medical associations have developed programs whereby patients can present grievances to committees of the respective associations; and

Whereas, These programs have provided an effective method of resolving real or fancied grievances of patients; therefore be it

Resolved, That this House of Delegates hereby commends those constituent state associations that have already established grievance committees to hear any complaints of the public and urges that all constituent associations adopt comparable programs and that the Secretary be instructed to transmit this resolution to all constituent medical associations.

Every county medical society was urged to co-operate actively in the organization and programs of health councils. They should also develop effective plans for handling medical calls.

The San Francisco session, held June 26-30, 1950, was one of the most successful in the history of the association. In addition to matters presented earlier in this report, the establishment of a student American Medical Association was authorized; reports on medical education and medical practice in England were adopted; some hospitals that make membership in specialty boards a requisite for appointment or advancement were criticized; and the continuation of the National Education Campaign was voted.

There was considerable discussion of a plan for the medical care of low-income veterans with nonservice-connected disabilities, but the plan was tabled. The consensus was that there should be more rigid rules for establishing the eligibility of a veteran with nonservice-connected disability for hospital, medical and surgical service.

One of the highlights of the meeting was the broadcast over two national networks of the inauguration of the president — Elmer Henderson. John W. Cline, of San Francisco, was chosen as president-elect.

I attended the Conference of County Medical Society Officers; the Conference of Presidents and Other Officers of State Medical Associations; and numerous other meetings, besides serving on the Reference Committee on Industrial Health.

The delegates from Massachusetts were gracious hosts at breakfast meetings of the New England delegation and matters of general interest were discussed.

An informal dinner of the delegates from states having only one or two delegates was very well attended. It was decided to entertain the officers and other delegates at some future session, and thus return the favors extended to us over many years. It was suggested that each state give small souvenirs, which would be emblematic of the state, or one of its products. All ideas that the members may have about this will be appreciated by me.

Dr. Brown moved that the report of the delegate to the American Medical Association be recorded in the minutes of the meeting.

This motion was duly seconded and was carried.

Dr. Brown then stated that the Committee on Officers' Reports suggested referring the American Medical Association proposal on the establishment

of county society control of Red Cross Blood Centers to the Blood Bank Committee of the Society for specific recommendations.

The resolution of the American Medical Association's House of Delegates concerning a grievance committee would be taken up when the amendments to the *By-Laws* were considered. The educational campaign against socialized medicine would also be taken up in connection with consideration of the budget.

Dr. Brown then moved that the proposal of the American Medical Association on the establishment of county-medical-society control of blood-bank centers be referred to the Blood Bank Committee of the Society.

This motion was duly seconded and was carried.

Mr. Hamilton R. Putnam then presented the report of the Executive Secretary and Public Relations Counsel, as follows:

On January 22, 1950, the House of Delegates of the New Hampshire Medical Society approved a plan combining in one executive office the duties and responsibilities of an executive secretary and public-relations counsel. Under this arrangement there was formulated and developed the following assignments under the direction and guidance of the Society's Executive Committee, operation of the executive office; conduct of educational campaign effort; development of public relations program; liaison and field representation; publicity; editing and publishing of the *Bulletin*; promotion of school essay contest; arrangements for commercial exhibits and annual meeting.

The brief summary that follows is respectfully submitted as a short, but concise, report showing the extent to which this society has become involved in a threefold program of public relations, educational campaign and executive secretaryship.

The executive office has become the focal point for public contact with the Society members and through which much correspondence flows to various committees and officers of the Society. Its staff provides varying services for physicians in the compilation of specific material and information, in the maintenance of files, packaging, mailing and mimeographing. Telephone and personal inquiries concerning Society business are directed to the executive office and through it to officers and committees. The Executive Secretary is directly responsible for the receipt, answering and movement of correspondence, maintenance of files, addressograph plates, mailing lists and the detailed administrative work of the Society. This has become an involved task in view of the scope of Society activities. During the six months' period January through June, 1950, the executive office received an average of 480 pieces of mail per month, 384 of which were first-class mail. Outgoing mail has averaged 634 pieces per month — 405 pieces first class. This has included individual mailing as requested by county society officers, committee members and officers, and correspondence associated with the school essay contest, exhibits, annual meeting and the *Bulletin*. During this same period the executive office received an average of 180 incoming telephone calls per month with outgoing calls of 135.

Educational campaign effort has been directed through various channels in 1950 with excellent results. This has included: distribution of educational campaign literature, public-speaking program, resolutions and contacts with the Congressional delegation.

The Society's 1950 campaign effort has been a broad continuation of its 1949 program with a two-pronged plan of public speaking and literature distribution. This effort resulted in the obtaining of 80 resolutions from service organizations, women's clubs, veterans groups, farm, civic and social clubs. These resolutions against Government-controlled medical care went directly to New Hampshire's Congressional delegation. Senators Bridges and Tobey and Congressmen Merrow and Cotton have received a grand total of approximately 12,000 individual communications from New Hampshire people as a direct result of the Society's educational campaign. More than 125,000 pieces of campaign literature have been distributed, much of it through and with the assistance of the Woman's Auxiliary in Nashua, Manchester, Concord, Franklin and in Cheshire, Strafford and Rockingham counties. Society representatives made 65 personal appearances during the period January through June. An average of 70 people were present at each meeting with the exception of a meeting in Rochester March 30 when the Society's executive secretary addressed 500 Grangers and guests, and at two meetings in Nashua February 6 when Dr. Ralph J. Gampell of the American Medical Association speaking staff addressed an estimated 1000 people. As a result of these meetings approximately 6000 heads of families in New Hampshire have heard about the national health issue through efforts of the Society. Under the direction of Dr. Deering G. Smith of Nashua, New England member on the American Medical Association Committee on Legislation, the Society's campaign manager has maintained a close contact with members of the New Hampshire Congressional delegation in Washington. Telegrams were sent Congressmen Merrow and Cotton on July 10 urging adoption of House Resolution 647 disapproving Reorganization Plan No. 27 to create a Department of Health, Education and Security. Both New Hampshire Congressmen were present and voted for the resolution, which was adopted 249 to 71.

Development of a public-relations program has shown marked progress in 1950. The Society enjoys a reasonably good press, and relations between the Society and news mediums remain close and co-operative. Newspapers and radio stations have been helpful during the year in assisting in the development of Society programs. Much of the public-relations progress, however, has come about through a greater appreciation by physicians and county-medical-society groups of the need for good press relations, the need for working together to solve local and community problems and the values to be found in allied professional relations. On March 3 the Society's executive committee met with representatives of the New Hampshire Accident and Health Underwriters Association. As a result considerable assistance has been provided by insurance men in furthering the Society's educational program. Many meetings have been held with legislative and lay groups. Greater public-relations progress will be possible through acceptance by the House of Delegates of the newly proposed "Code of Co-operation," which has been drawn up through a series of conferences between representatives of the Society, the press and radio stations.

The Society's executive secretary and public-relations counsel has provided liaison service and field representation to county medical society officers and to other committees as well as the executive committee. This has included periodic visits to hospitals, newspapers, radio stations, state agencies and departments, insurance representatives, lay groups and individual physicians with an aim toward furthering the Society's programs.

Publicity releases issued by the Society during the period January through June have totaled slightly more than 1100 column inches of published copy appearing in New Hampshire weekly and daily newspapers. This publicity has been associated with the Society's educational campaign and press announcements of plans and programs carried on by the Society.

The Society's *Bulletin* has become rather widely read by physicians throughout the State, by a group of lay people to whom it is sent and by newspaper and radio people. Purpose of this bulletin is to provide members of the profession and allied professional groups with information about the Society and to encourage doctors to carry out programs adopted by the Society. Bulletins were published in March and June.

The Society's school essay contest drew 57 entries from private, parochial and public schools throughout the State. All details in connection with this affair were handled by the executive office. The Society provided $50 in cash for prizes. Contest winner was Miss Alice Rouleau, a senior at Spaulding High School in Rochester. On behalf of the

Society the Executive Secretary invited the contest winner and her mother to a luncheon in Concord and later arranged for the Governor to make the cash presentation to the winner.

Arranging for commercial exhibits and the annual meeting has taken a great deal of time of the executive office staff. This has included planning the exhibits, selling space, communicating with exhibitors, arranging details, planning for the annual meeting, assignment of meeting rooms, printing and distribution of programs, committee reports and so forth. Several meetings have been held during the period with the hotel management in order to work out details of the convention. Meetings have also been held with Mr. Getty Page, of the Vermont State Medical Society, and with the committee members and officers.

During the first quarter of 1950, January, February and March, personnel of the executive office provided 1600 man-hours of service to the Society. During the second quarter, April, May and June, man-hours provided amounted to 1377. This total six months' service of 2977 man-hours was provided at a cost to the Society of $1.83 per hour for services of personnel of the executive office.

Dr. Brown, for the Committee on Officers' Reports, expressed high approval of the report of the Executive Secretary and Public Relations Counsel and commended the office of the Executive Secretary for the vast amount of minute detail that it has had to deal with and has so successfully covered in preparation for this meeting. He moved that the report of the Executive Secretary be accepted as printed.

This motion was duly seconded and was carried.

President Bowler then presented the report of the Council of the New England State Medical Societies as follows:

During the fiscal year of the Council the public-relations program discussed in an open regional meeting in 1949 has been augmented at the state levels, and the best thinking and planning of the various medical societies has been exchanged in Council sessions to the advantage of the region.

The Council had an interesting meeting last November at which the problem of hospital care for the chronically ill, the question of hospital-physician relationships and the matter of effective medical public-relations were discussed.

It was with regret that the officers canceled the proposed reception and dinner in honor of the Congressional delegation from New England and the editors of the leading newspapers of our states. The absence from the area of so many of the Congressmen who were engaged in committee studies of national interest forced the decision to postpone the testimonial to another year. Certainly the gesture planned by the New England Medical Societies was appreciated by many of those who were to have been our guests, and who wrote splendid letters to your officers.

The recent meeting on national health legislation demonstrated anew the great value of the Council as an organizing group to carry forward an educational program for our physicians on matters of mutual interest. Approximately 150 physicians and their guests attended this important session, and each was furnished with a printed digest of the six major national health acts discussed. The evening was climaxed by an interesting address by Dr. Elmer L. Henderson, of Louisville, Kentucky, president-elect of the American Medical Association, on the national education campaign of the Association.

The Council has set a pattern that may well be copied elsewhere in regional planning. In the past five years a wide range of topics have been discussed, and our state societies have benefited thereby. In this time we have seen each of the New England states establish a central executive office with full-time personnel manning it.

John E. Farrell, Sc.D., who has served as executive secretary-treasurer for five years, and whose enthusiasm and energy has kept the Council operating efficiently, wished to relinquish the office. His desires were acceded to with regret, and Creighton Barker, M.D., was chosen to succeed him.

It is hoped that the New Hampshire Medical Society will continue as a member of the Council and that an appropriation of $100 will be made as its share of the expenses.

Dr. Brown, for the Committee on Officers' Reports, moved that the report of the Council of the New England State Medical Societies be included in the minutes of the meeting.

This motion was duly seconded and was carried.

The reports of the councilors were then presented, as follows:

Belknap County

The Belknap County Medical Society is one of the smaller county societies in the state, but shows a healthy growth of from 40 members in 1948 to 43 in 1949.

In November, 1949, our society was greatly saddened when one of our younger and most loved physicians, Dr. Karl L. Smith, died suddenly from coronary occlusion, in his thirty-ninth year.

The Society has held seven meetings during the year, the meetings being held on the second Tuesday of each month. I think it is worthy of note that although it is one of the smaller county units, our society holds more regular meetings than any other county society in the State.

The first meeting of the year was held at the Winnisquam House on October 11, 1949, with 36 members and guests present. An excellent paper was given by Dr. M. Strauss, acting chief of the Cushing Veterans Administration Hospital, on "Lower-Nephron Nephrosis."

The November 3 meeting was held at the Laconia Hospital, with 30 members, and guests present. Dr. Thannhauser, of the Pratt Diagnostic Clinic, gave an enlightening discussion on several cases presented.

The December 13 meeting was held at the Winnisquam House. Dr. Deering Smith, our state secretary and treasurer, was the speaker and he gave an informal talk on the problems of the New Hampshire Medical Society.

The January 10, 1950, meeting was held at the Arlberg Inn, with 30 members and guests present. Our state president, Dr. John Bowler, of Hanover, gave an address emphasizing the problems of the national and state medical societies.

The February 14 meeting was held at Garnet Inn, Center Harbor, with 33 members present. Dr. Shields Warren, of Boston, presented an excellent paper on "Surgery of the Peptic Ulcer."

The March 14 meeting was held at the Winnisquam House, and Dr. J. Sidney Stillman, Chief of the Medical Service of the Robert Breck Brigham Hospital in Roxbury, Massachusetts, spoke on "Rheumatoid Arthritis and ACTH," and other therapeutic agents of this most interesting and difficult disease.

The April 27 meeting was held at the Winnisquam House, with thirty-seven members present. The meeting followed a clinic on "Heart Disease in Children" in the afternoon, and the discussion was opened and closed by Dr. Eley and Dr. Longino, of the Children's Hospital in Boston.

C. L. Smart

Grafton County

The usual program of spring and fall meetings has been carried out. The autumn meeting was held at Hale's Tavern, Wells River, Vermont; and the spring meeting at Hotel Coolidge in White River Junction, Vermont. Both meetings were held in the evening, preceded by a social hour and dinner. The attendance was good, and an interesting program was presented. Several new members have been elected during the current year.

Arthur W. Burnham

Hillsborough County

The forty-fifth annual meeting of the Hillsborough County Medical Society was held at the Nashua Country Club on November 22, 1949. One hundred and forty-eight members and their wives attended the dinner. Following the dinner Dr. Dennette Adams, of Boston, spoke on "Chronic Headaches." His talk was extremely interesting and provoked considerable discussion.

Dr. John Bowler, president of the New Hampshire Medical Society, spoke on the current situation of the medical profession in the State.

The Secretary suggested that a new program committee be named to conduct the next Hillsborough County Medical Society meeting in the Spring of 1950. The committee was named by Dr. Backus and consists of Dr. Goldkamp, Dr. Deitch and Dr. Flagg. A committee on cancer was nominated and elected and consists of Dr. Biron, Dr. Umpa and Dr. Loverud.

The meeting then took up discussion of the Cheshire County Plan, and many of the doctors spoke pro and con in connection with this matter. A motion was made that the Society go on record as opposing the Cheshire Plan and the vote was carried.

Six new members were elected to membership in the Hillsborough County Medical Society: Dr. G. L. Bastian, Jr., Dr. J. Hebert, Dr. G. S. Baldry, Dr. L. A. Laflamme, Dr. J. F. Macek and Dr. E. O. Warren.

The following were elected as honorary members: Dr. G. B. Hoitt, Dr. H. A. Streeter, and Dr. L. T. Togus.

The election of officers then took place and the following members were elected:

President	Dr. Norman Crisp	
Vice-President	Dr. Robert Biron	
Secretary-Treasurer	Dr. Lloyd L. Wells	
Board of Censors	Dr. Daniel Sullivan	
House of Delegates	Dr. Raymond Marcotte	2 yr.
	Dr. Frank Flagg	2 yr.
	Dr. Edward D. Hagerty	2 yr.
	Dr. Oscar Burns	1 yr.
Educational Committee	Dr. Daniel Sullivan	
Jurisprudence Committee	Dr. David Parker	

A rising vote of thanks was given Dr. Dennette Adams for his excellent talk and the meeting finally adjourned.

A special meeting of the Hillsborough County Medical Society was held at the Crowley School Auditorium, Lake Street, Nashua, on Sunday, March 19, 1950, Dr. Norman Crisp, presiding.

The object of this meeting was to consider the facts in the case of Dr. Hermann Sander relative to the Hillsborough County Medical Society.

The Constitution and By-Laws of the Hillsborough County Medical Society were reviewed and clarified in regard to the proper procedure of filing charges against a member of the Society. In accordance with the Constitution: — "Charges against a member must be made in writing and be delivered to the Secretary, who shall immediately furnish a copy to the accused and to the Chairman of the Board of Censors. The Board of Censors shall investigate the charges on their merits, but no action shall be taken by the Board within ten days of the presentation of the charges to the accused, nor before giving the accused and accusers ample opportunity to be heard." "No action shall be taken by the Society in such case until at least six weeks have elapsed since the filing of the charges."

The meeting was adjourned pending the filing of charges and the report of the Board of Censors.

The following official statement was released to the press: "The Hillsborough County Medical Society considered the case of Dr. Hermann N. Sander in relation to the County Medical Society. No other business was taken up. No comment will be made about this consideration."

A special meeting of the Hillsborough County Medical Society was held at the Masonic Temple, Milford, on Tuesday, May 9, 1950. One hundred members attended this meeting, which was called to order by the president, Dr. Crisp, at 8 p.m.

The object of this meeting was to hear and consider the report of the Board of Censors relative to the case of Dr. Hermann N. Sander.

The majority report was as follows: "The undersigned members of the Board of Censors of the Hillsborough County Medical Society find that a charge of gross misconduct against Dr. Hermann N. Sander by reason of his injection of air into the body of Abbie Borroto — admittedly an irrational act, is sustained." "We therefore recommend that he be expelled from this Society."

The minority report was as follows: "As a member of the Board of Censors of the Hillsborough County Medical Society, I concur in finding that the charge against Dr. Hermann N. Sander of gross misconduct as a physician by reason of injection of air into the body of Abbie Borroto, an admittedly irrational act, is sustained. I therefore recommend that he be suspended from the Society for a period of six months from this date and then reinstated to membership if licensed to practice medicine in New Hampshire at that time."

A motion was made that the majority report be accepted.

After a long discussion including substitute resolution, it was ruled that the report of the Board of Censors must be acted on as recommended to this Society and as made in the first motion. The motion was then voted on with 64 yes, 35 no, in favor of accepting the majority report of the Board of Censors. As the Constitution and By-Laws requires that three fourths of the members voting are necessary to expel a member from the Society the recommendation of the majority report was not sustained. A motion was then made and seconded that the secretary of the Society write to Dr. Sander stating that he was no longer a member of the Society. The motion was then passed and voted upon as follows: 80 yes, 20 no.

The following official statement was released to the press: "At a meeting of the Hillsborough County Medical Society it was voted that Doctor Hermann N. Sander is no longer a member of this Society."

T. F. Rock

Coos County

In the Fall of 1949 the semiannual meeting of the Society was held at the Glen House, with Dr. John P. Bowler as guest speaker.

The Spring meeting was held at the McKee Inn. Dr. Donald Munro, noted brain specialist of Boston, was in town at the time, and we were very fortunate in being able to have him as speaker. A dinner was served after which we listened to an interesting talk on the care of paraplegic patients. Later an address was given, to which the public was invited, and at this time colored motion pictures were shown, which added to the information that Dr. Munro had given.

Our society meetings are semiannual unless there is reason to call a special meeting.

W. M. Bronson

Merrimack County

On January 5, 1949, a meeting was held at Alumni House, Concord. Dr. C. Stuart Welch, of Tufts College Medical School, gave an illustrated lecture on "Ulcerative Colitis." General discussion of the American Medical Association $25 special assessment followed.

On April 6, 1949, a joint meeting with the Auxiliary was held at the Winnisquam House, Tilton. Discussion of the American Medical Association education campaign followed, as well as discussion of methods of securing doctors to enlist for active military service. Discussion of means of increasing interest in voluntary health and accident hospitality plans such as Blue Cross and Blue Shield was also presented. Dr. Charles Roth, Boston, gave an illustrated lecture on diseases of the leukocytes.

On June 29, 1949, a meeting was held at Mojalaki Club, Franklin. Dr. Woodman gave a report on proceedings of the House of Delegates at the Wentworth Hotel, Newcastle.

On October 5, 1949, at a meeting at the Mountain View House, Warner, Dr. John P. Bowler, and Dr. Deering G. Smith, president and secretary of the New Hampshire Medical Society, discussed various Society problems. Mr. Angus Woodbury spoke, representing Mr. Hamilton

Putnam. Dr. John S. Lyle, Hitchcock Clinic, spoke on the use of Dicumarol in prophylaxis of postoperative embolism.

On January 5, 1950, at a meeting at Harvey Lake Inn, Northwood, Judge Thornton Lorimer spoke on legal aspects of medicine. Mr. Hamilton Putnam discussed the problems of the New Hampshire Medical Society.

On April 5, 1950, at a meeting with the Auxiliary at Angelo's restaurant, Concord, Dr. George Morris, Boston, spoke on "Industrial Dermatitis." There was a general discussion of the "Cheshire Plan" and also of Blue Cross and Blue Shield.

On May 24, 1950, a meeting at Daniel Webster Inn, Franklin, was given over largely to Blue Cross and Blue Shield matters.

HENRY H. AMSDEN

CARROLL COUNTY

Stated meetings of the Carroll County Medical Society usually consist of one meeting in the early part of June. This meeting was held on June 14 at which time we had election of officers. Charles Smith, Conway, was elected president; Louise M. Paul, Wakefield, vice-president; W. J. Paul Dye, Wolfeboro, secretary and treasurer; Dr. Dye and Francis J. C. Dube, Center Ossipee, delegates to the New Hampshire Medical Society; Howard P. Sawyer, Brookfield, alternate delegate.

This meeting was held in conjunction with the staff meeting of Huggins Hospital. Two new members were elected to the Society: Dr. Spring, of Conway, and Dr. Paulson, of Moultonboro.

The reason for the infrequent meetings of the Medical Society is that it has been impossible to get the men from the northern part of the county, with the exception of Dr. Smith, to attend the meetings of the Society. We are at a loss to know what to do to get the men in the northern part of the county to co-operate with the rest of us. Since 9 of the 16 members of the Society are on the staff of Huggins Hospital, Society affairs are talked over more or less at the weekly staff meetings of the hospital. If the men from the northern part of the county would attend the meetings we should be very glad to have stated county meetings in their neighborhood.

R. W. TUTTLE

STRAFFORD COUNTY

The Strafford County Medical Society has had two regular meetings within the year — one October 26, 1949, and the other on May 3, 1950. At the time of the first meeting Dr. Feldman, of the Arthritic Commission of Boston, discussed in considerable detail the ACTH factor in the treatment of rheumatoid arthritis. The meeting was well attended and a prolonged period of questioning and discussion followed.

At the second meeting Dr. Richard Overholt, of Boston, described various phases of thoracic surgery, his talk being confined to surgical interference in the various fields of lung pathology, particularly tuberculosis, bronchiectasis and carcinoma. This meeting was also well attended and the discussion that followed was extremely informative.

At this writing we have 42 active members and 6 life members. The meetings have been exceedingly well attended, and the delegates have been very active in attendance at the various special and state meetings.

BERNARD J. MANNING

ROCKINGHAM COUNTY

At the spring meeting in April, 1949, the following events took place: letters were read from Senator Bridges and from Senator Tobey, stating that both were opposed to Government medicine; three new members were accepted for the county society (these were Drs. Land, Randall and Strauss); the appointment of the Rockingham County Education Campaign Organization was made public; a resolution was adopted by the members of the Rockingham County Medical Society that put this society on record as being against compulsory health insurance; and in the afternoon session a speech by Clem Whitaker was presented by a wire recording — after this there was discussion by Mr. Putnam of the progress and plans for the New Hampshire campaign.

At the fall meeting in 1949 a new slate of officers was presented and accepted: president, James Sanders; vice-president, Oscar Gilbert; secretary-treasurer, Donald W. Leonard; board of censors, Wayne Bryer, Willard Montgomery and Anthony E. Peters. Cleon Colby was made an honorary member of the Rockingham County Medical Society. Dr. Bowler reported that the F.B.I. was investigating local medical societies. At the afternoon session Dr. Hans Waine gave an excellent talk entitled, "Recent Advances in Rheumatology."

At the spring meeting in 1950 Dr. Wallis Walker was elected an honorary member of the Society. Three new members were accepted by the Society: Drs. Conroy, Fidler and Hunter. There was discussion about combining the financial drives in this state of the Heart Association and the Arthritis Association. There was discussion about the methods by which the doctors of the Society could help the County Commissioners in taking care of needy children who needed tonsillectomies. Dr. F. S. Gray was elected as the Rockingham County member of the Blue Shield Co-ordinating Committee.

In February, 1950, the society sustained a great loss in the death of Donald W. Leonard, our secretary for many years, and with him died the *Rockingham County News*, which he published and which was as great an asset to the society as the publisher.

Dr. Henry Saltonstall, of Exeter, was elected to serve as secretary in Dr. Leonard's place.

FREDERICK S. GRAY

CHESHIRE COUNTY

We had a special meeting on March 3, 1949, for the purpose of co-operating with the American Medical Association against socialized medicine. A committee of A. C. Johnston, L. A. Ford, J. P. MacAllister and W. H. Lacey was appointed. We took an active part in the campaign and presented our Cheshire County plan at the last June meeting of the New Hampshire Medical Society.

On May 19, 1949, our regular annual meeting, we set up a public-relations committee of Cheshire County Medical Society and heard an excellent address by Dr. Fromer, dermatologist of the Lahey Clinic.

On October 6, 1949, we had our semiannual meeting and as our guests had the President, Secretary-Treasurer, Mr. Comee of the New Hampshire–Vermont Services, and Mr. Putnam, our executive secretary.

On April 26 of this year we had our annual meeting, with election of the officers as now constituted.

We lost 2 members by death and 1 by transference, but gained 3 new ones.

ALBERT C. JOHNSTON

Dr. Brown moved that the reports of the Councilors be accepted.

This motion was duly seconded and was carried.

(To be continued)

The New England Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Official Organ of
THE MASSACHUSETTS MEDICAL SOCIETY
and
THE NEW HAMPSHIRE MEDICAL SOCIETY

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*Representing the New Hampshire Medical Society.

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MATERIAL, other than original articles, should be received not later than noon on Thursday, three weeks before date of publication.

THE JOURNAL does not hold itself responsible for statements made by any contributor.

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ROADBLOCKS TO CLINICAL RESEARCH

HOSPITAL records are grist for the clinical-research mill and provide food for growth of both therapeutic and preventive medicine. At the present time the files of hospitals affiliated with New England medical schools are literally bulging with the patiently accumulated information assembled by many observant minds over many productive years. Yet that information is often so loosely catalogued, cross-referenced and integrated that the mere assemblage of data takes weeks and months instead of days and weeks. For example, the analysis of a hundred cases of poliomyelitis or of patients treated surgically for thrombophlebitis or of the results of cesarean births constitutes a formidable undertaking, particularly when the data sought lie buried in diverse hospitals in their several departments of surgery, medicine, obstetrics, orthopedics, roentgenology and pathology. The investigator expends the greatest part of his effort during precious hours of freedom as a medical Martin Chuzzlewit copying manuscripts that should be as easily reproduced as V Mail. It is not the wasted energy that is to be deplored so much as the lost time. To consume a year accumulating information that should be available in a month is to slow the progress of medicine more than twelvefold. A single medical question successfully answered raises so many others that the process of research characteristically moves forward in geometric increments.

As it is now, the physician undertaking some phase of clinical research leaves the record room looking like a newsboy when the afternoon editions first come in. As likely as not he has become *persona non grata* with the file clerks, for whom he creates work and confusion when not operating along familiar administrative channels. If the hospital permits, the records will be heaped in a corner for months on end while the future Galen copies and recopies, arranges and rearranges clinical data and tables in order to test the hypothesis he has formulated. Even in the well run hospitals that have a reading room or alcove expressly set aside for research the problem of processing record heaps remains the same.

Much needed are modern technical facilities for microfilming and photostating designed to extract portions of the hospital record for analysis or synthesis according to the investigator's decision. Likewise, in many hospitals there is still a crying need for central filing and better cross-indexing of departmental data. The high cost of inadequate systems is to be measured in lost time rather than wasted labor.

ALL DRUGS ARE DANGEROUS

THE paper by Smith, Jones and Cochran, published elsewhere in this issue of the *Journal*, calls attention once more to the possibility that any chemical agent with the property of altering the physiologic processes of the body may have also toxic effects when taken in large quantities or by sensitive persons.

The case reported is that of a seventeen-month-old girl who inadvertently ingested an unknown number of ferrous sulfate tablets, which had been left within the range of her activities. She had been brought to the hospital cyanotic and unresponsive, vomiting and diarrhea having developed about four hours after the tablets were swallowed. Despite emergency measures consisting of the injection of Coramine intravenously, the transfusion of plasma, oxygen inhalation and the administration of methylene blue to combat a possible methemoglobinemia, vomiting of bloody fluid and the passage of black, watery stools continued, and she died approximately seven hours after the first symptoms were noted.

A review of the literature shows that while poisoning from compounds of iron is not common, enough cases have occurred to justify a warning in respect to their use. From 1850 to 1890 several cases of poisoning from iron compounds apparently occurred, severe gastrointestinal irritation making up the characteristic picture. In the present century several additional cases have been added, some of which were fatal.

The case reported is of especial value from a scientific point of view because of the thorough investigation that it received, including particularly the complete autopsy report. This justifies the conclusion that the main point of attack of an overdose of ferrous sulfate is the gastrointestinal mucosa, where an intense irritation is succeeded by areas of necrosis. Further than this, masses of iron pigment were discovered in the portal veins, the liver parenchyma cells showed degenerative changes, and the lungs were edematous and hemorrhagic.

The matter of treatment presents a problem, since BAL seems to enhance the toxicity of iron, rather than neutralizing it. General supportive measures and transfusion are indicated, with administration of methylene blue if methemoglobinemia proves to be a regular part of the picture. First aid should consist of the usual emesis and feeding of raw eggs or milk, the protein of which could at least partially absorb the iron.

In these cases the usual ounce of prevention is worth any amount of cure, and their prevention can be effected only by thorough indoctrination of the public with the danger of leaving any drug, no matter how apparently innocuous, within the reach of children.

NEXT TO GODLINESS

In England now, if one correctly interprets a peripatetic correspondent of *The Lancet*,* cleanliness, even if it still ranks next to godliness, comes dearer. The discussion is initiated by the comment of a Danish visitor on the dirtiness of the food and centers on the difficulty of attaining, under certain circumstances, even such a rudimentary level of ordinary decency as washing the hands after the toilet ritual.

This correspondent, a woman, found that a penny was necessary to gain admission to the public lavatory in Leicester Square and that threepence was required for the use of a towel after washing. Men, she suspects, although they are frequently charged not even the penny, do not often bother with the ablutions, the desirability of which should seem so obvious, but perhaps she has a grudge against men.

Soap is becoming less scarce in England, water is still available for necessary functions, and it would seem that the Ministry of Health, if none other, might engineer an opportunity for the fastidious to use a paper towel at something under threepence a wipe.

There is no question that an accurate inquiry into the post-excretory habits of Americans, with so many opportunities for cleanliness freely available, would be equally disconcerting. When the inescapable surmise is applied to restaurant personnel the prospect of dining at home seems doubly attractive.

DIABETES WEEK

This year Diabetes Week, the climax of the diabetes-detection drive sponsored by the American Diabetes Association, will begin on November 12. The drive constitutes an effort, led by physicians and supported by business and industrial leaders in the community, to disclose the estimated 1,000,000 undiscovered cases of diabetes in

*In England now. *Lancet* 1:1130, 1950.

the country. The campaign last year uncovered 7500 "hidden" cases; with the American Medical Association's approval of self-testing and with wider co-operation from business and industry, it is hoped that the current drive will reveal even more cases. During the week doctors in many communities in Massachusetts and other states are offering free tests of the urine.

The campaign merits the wholehearted support of all members of the medical profession. The goal of discovering the patients with unrecognized diabetes, so that they can be referred for treatment, represents preventive medicine at its best: case finding and constructive efforts to control a disease that is still one of the leading causes of death.

We find in one of the Journals, an article [sic] on the art of coughing. It is deemed unnecessary to give the tactics in ours.

Boston M. & S. J., *October 23, 1850*

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

The November schedule for Consultation Clinics for Crippled Children in Massachusetts under the provisions of the Social Security Act follows:

Orthopedic Clinics	Date	Clinic Consultant
Haverhill	November 1	William T. Green
Greenfield	November 2	Charles L. Sturdevant
Lowell	November 3	Albert H. Brewster
Salem	November 6	Paul W. Hugenberger
Brockton	November 9	George W. Van Gorder
Gardner	November 14	Carter R. Rowe
Worcester	November 17	John W. O'Meara
Springfield	November 21	Garry deN. Hough, Jr.
Fall River	November 27	David S. Grice
Hyannis	November 30	Paul L. Norton

Rheumatic Fever Clinics	Dates
Fitchburg	November 1, 8, 15, 22, 29
North Reading	November 7, 14, 21, 28

Plastic Clinics	Consultant
Eastern Massachusetts	Bradford Cannon
	Semi-monthly
Western Massachusetts	Joseph M. Baker
	Monthly

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

NOTICES

ANNOUNCEMENT

Dr. Richard W. Thaler announces the opening of his office for the practice of internal medicine at 9 Endicott Street, Beverly.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

The following regular meetings at the Joseph H. Pratt Diagnostic Hospital have been scheduled:

Medical Clinicopathological Conference. First Wednesday of every month, 9–10 a.m. Dr. H. Edward MacMahon.

Pediatric Pathological Conference. Third Wednesday of every month, 9–10 a.m. Dr. H. Edward MacMahon.

Medical Grand Rounds. Weekly on Thursdays, 9–11 a.m.

Medical Conference with guest speaker. Weekly on Fridays, 9–10 a.m.

Surgical Pathological Conference. Second and fourth Fridays of every month, 12 noon to 1:30 p.m.

Therapeutic Conference. Second and fourth Fridays of every month, 2–4 p.m. Robert P. McCombs, M.D., moderator.

X-Ray Conference. Second and fourth Fridays of every month, 4–6 p.m. Dr. Merrill C. Sosman.

Surgical Grand Rounds. Weekly on Saturdays, 9–10 a.m.

SPRINGFIELD HOSPITAL

The educational program of the Springfield Hospital for the month of November is as follows:

Wednesday, November 1. 12 m. Pathology Conference in the Staff Conference Room on the subject "Malignancies of the Skin." Dr. William Kaufmann.

Saturday, November 4. 10 a.m Radiology Conference in the Staff Conference Room on the subject "Diagnostic X-Ray Problems of Special Interest." Dr. J. Turner. 11 a.m. Combined Medical-Surgical Grand Rounds in Staff Conference Room.

Wednesday, November 8. 12 m. Conference on psychosomatic medicine in the Staff Conference Room, the subject being "Basic Concepts in Analytical Psychiatry." Dr. Calvert Stein.

Thursday, November 9. 12:15 p.m. Staff meeting in the Staff Room.

Saturday, November 11. 10 a.m. Seminar on Surgical Technic — First Session in the Staff Conference Room — Dr. F. W. Hoyt and staff. 11 a.m. Clinicopathological Conference in the Staff Conference Room.

Wednesday, November 15. 12 m. Pathology Conference in the Staff Conference Room on the subject "Lesions of the Male Genital Tract." Dr. William Kaufmann.

Saturday, November 18. 11 a.m. Medical Death Review Meeting in the Staff Conference Room.

Tuesday, November 21. 8:30 p.m. Guest Speaker Conference in the Staff Conference Room on the subject "Methods of Producing Anesthesia for Intra-Abdominal Surgery." Dr. Ralph M. Tovell, chief anesthesiologist, Hartford Hospital, Hartford, Connecticut.

Wednesday, November 22. 12 m. Orthopedic Conference in the Staff Conference Room. Case Presentation by Dr. P. Carson.

Saturday, November 25. 10 a.m. Seminar on Surgical Technic in the Staff Conference Room. Dr. F. W. Hoyt and Staff. (Second Session.) 11 a.m. Surgical Death Review Meeting in the Staff Conference Room.

Wednesday, November 29. 12 m. Neurosurgical Conference in the Staff Conference Room. Case Presentation by Dr. Joseph Hahn.

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

A meeting of the Massachusetts Society of Examining Physicians will be held at the Harvard Club of Boston on Wednesday, November 15, at 6:30 p.m. After the dinner Dr. Walter L. Cronin, civil-defense director of the City of Cambridge and consultant to the National Securities Resources Board, will speak on the subject "Casualty Management and Civil Defense Incident to Modern Warfare." Dr. Cronin will also show some films in conjunction with his talk.

Some new films will be shown by Dr. Donald E. Currier, surgeon general of the Commonwealth of Massachusetts, and there will be a short talk on the subject by Dr. Charles H. Bradford, member of the Staff of the Boston City Hospital, and Dr. William J. Brickley, medical examiner of the Northern District of Suffolk County.

Everyone interested in the subject is cordially invited to attend.

HARTFORD MEDICAL SOCIETY

The Program Committee of the Hartford Medical Society announces the last three of the fall series of lectures to be presented in the Hunt Memorial Building, Hartford, Connecticut, on the dates indicated, at 8:30 p.m.:

November 20. Blood Dyscrasias. Dr. Cyrus C. Sturgis.

December 4. Experiences with Radical Surgery in Advanced Cancer of Female Genital Tract. Dr. Alexander Brunschwig.

December 18. Causes and Control of Arteriosclerosis. Dr. William Dock.

NEW ENGLAND CARDIOVASCULAR SOCIETY

The New England Cardiovascular Society announces that its 1950–1951 series of meetings will be held at the New England Mutual Hall, 225 Clarendon Street, Boston. This change has been necessitated by the increasing membership in the Society and attendance at the meetings, which can no longer be comfortably accommodated in the hospitals and medical schools. This does not apply to the annual meeting, which will be held in the Boston Medical Library as usual. The scientific program for 1950–1951 will be as follows:

November 27, 1950. Massachusetts General Hospital. Edward F. Bland, M.D., chairman.

December 18, 1950. Peter Bent Brigham Hospital. Samuel A. Levine, M.D., chairman.

January 15, 1951. Boston City Hospital. Laurence B. Ellis, M.D., chairman.

February 19, 1951. Massachusetts Memorial Hospitals. Robert W. Wilkins, M.D., chairman.

March 19, 1951. Beth Israel Hospital. Herrman L. Blumgart, M.D., chairman.

April 23, 1951. Children's Medical Center. Benedict F. Massell, M.D., chairman.

May 21, 1951. New England Center Hospital. Samuel H. Proger, M.D., chairman.

June 4, 1951. Annual meeting. Boston Medical Library.

AMERICAN FEDERATION FOR CLINICAL RESEARCH

The Eastern Sectional Meeting of the American Federation for Clinical Research will be held at Walter Reed General Hospital, Washington, D. C., on December 9.

Abstracts of papers to be presented should be sent to Benjamin Manchester, M.D., 3200-16th Street, N. W., Washington, D. C., prior to November 1.

PAN-PACIFIC SURGICAL ASSOCIATION

The Fifth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, November 10-21, 1951.

The object of the Pan-Pacific Surgical Association is to bring together surgeons from countries bordering on the Pacific Ocean so as to permit the exchange of surgical ideas and methods and to develop a spirit of good fellowship among the various races represented. Although it was planned that meetings would be held every three years, world events have permitted but four conferences since the organization was founded — the first in 1929 and the last in 1948.

The Fifth Congress provides an opportunity for doctors to combine a delightful vacation in Hawaii with attendance at a scientific meeting, the program of which will be presented by leading surgeons from countries of the Pacific area. Doctors are urged to bring their families with them and are promised luxurious accommodations.

Dr. F. J. Pinkerton, president of the Association, has been officially appointed as travel agent for those coming to the meeting. To be assured of preferred accommodations, travel and hotel reservations should be made through Dr. Pinkerton.

Further information may be obtained from the office of the Pan-Pacific Surgical Association, Suite 7, Young Building, Honolulu, T. H.

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The first fall meeting of the New England Society of Physical Medicine will be held at the Hotel Kenmore, Boston, on Wednesday, November 8, at 8 p.m. The subject of the meeting will be "Recent Advances in Physical Medicine: A report of progress." Drs. Jacob Rudd, Fritz Friedland, Louis Feldman, John Bisgrove and John Doyle will lead the discussion.

Physicians, physical therapists, occupational therapists and correctional therapists are invited to attend.

SOCIETY MEETINGS AND CONFERENCES

October 28. Massachusetts Association of Medical Technologists. Page 324, issue of August 24.
October 30–November 3. American Public Health Association. Page 240, issue of August 10.
October 31. Hampden District Medical Society. Page 629, issue of October 19.
October 31. Boston City Hospital House Officers' Association. Page 582, issue of October 12.
October 31–November 3. International College of Surgeons. Page 170, issue of July 27.
November 1. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.
November 1. Hampshire District Medical Society. Page 170, issue of July 27.
November 1. New England Pediatric Society. Page 582, issue of October 12.
November 1–29. Springfield Hospital. Page 679.
November 5 and 6. American Society for the Study of Arteriosclerosis. Page 170, issue of July 27.
November 6–8. New England Postgraduate Assembly. Hotel Statler, Boston.
November 6–9. International Medical Assembly. Page 548, issue of October 5.
November 8. New England Society of Physical Medicine. Notice above.
November 9. The Psychoneurotic — A Pest or a Patient. Dr. Jackson Thomas. Pentucket Association of Physicians. 8:30 p.m. Haverhill.
November 9–11. Association of Military Surgeons of the United States. Page 582, issue of October 12.
November 10. American Trudeau Society. Page 280, issue of August 17.
November 14. New England Society of Anesthesiologists. Page 512, issue of September 28.
November 15. Massachusetts Society of Examining Physicians. Page 679.
November 20. Hartford Medical Society. Notice above.
November 27–June 4. New England Cardiovascular Society. Notice above.
November 27. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.
November 28. Norfolk District Medical Society. Page 629, issue of October 19.
December 4. Hartford Medical Society. Notice above.
December 7–9. New York State Society of Anesthesiologists. Page 582, issue of October 12.
December 9. American Federation for Clinical Research. Notice above.
December 18. Hartford Medical Society. Notice above.
January 22. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.
January 23. Norfolk District Medical Society. Page 629, issue of October 19.
January 31. American Academy of Orthopaedic Surgery (see Kappa Delta Award for Research in Orthopedic Surgery). Page 736, issue of May 4.
February 13. New England Society of Anesthesiologists. Page 512, issue of September 28.
February 27. Norfolk District Medical Society. Page 629, issue of October 19.
March 26. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.
March 27. Norfolk District Medical Society. Page 629, issue of October 19.
April 9–13. American College of Physicians. Page 296, issue of June 15.
April 10. New England Society of Anesthesiologists. Page 512, issue of September 28.
April 24. Norfolk District Medical Society. Page 629, issue of October 19.

District Medical Societies

HAMPDEN

October 31. 6:00 p.m. Hotel Kimball, Springfield.

HAMPSHIRE

November 1. 4:30 p.m. Northampton State Hospital.
January 3. 4:30 p.m. Veterans Administration Hospital, Northampton.
March 7. 4:30 p.m. Cooley-Dickinson Hospital, Northampton.
May 2. 4:30 p.m. Annual Meeting. Jones Library, Amherst.

NORFOLK

November 28.
January 23.
February 27.
March 27.
April 24.
May 9. Annual Dinner.

All meetings except the Annual Dinner will be held at the Boston Medical Library, 8 Fenway, at 8:00 p.m.

(Notices concluded on page xvii)

NOTICES *(Concluded from page 680)*

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, NOVEMBER 2

THURSDAY, NOVEMBER 2

*9:00-10.00 a.m. Cardiac Grand Rounds. Main Amphitheater, Children's Medical Center.

*9:00-11:00 a.m. Medical Grand Rounds. Joseph H. Pratt Diagnostic Hospital.

FRIDAY NOVEMBER 3

*9:00-10.00 a.m. Medical Conference. Joseph H. Pratt Diagnostic Hospital.

*9:00 a.m.-12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

*10:00 a.m. Surgical Clinic. South End Health Unit, 57 East Concord Street, Boston.

*1:30 p.m. Tumor Clinic. Out-Patient Department, Mount Auburn Hospital, Cambridge.

SATURDAY, NOVEMBER 4

*9:00-10:00 a.m. Surgical Grand Rounds. Joseph H. Pratt Diagnost'c Hospital.

MONDAY, NOVEMBER 6

*11:30 a.m.-12:15 p.m. Clinical Conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.

*12:15-1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

TUESDAY, NOVEMBER 7

*12:15-1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30-2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachustts General Hospital.

WEDNESDAY, NOVEMBER 8

*12:00 m. Clinicopathological Conference. Margaret Jewett Hall, Mount Auburn Hospital, Cambridge.

*12:00 m.-1:00 p.m. Clinicopathological Conference. (Children's Medical Center.) Amphitheater, Peter Bent Brigham Hospital.

*8:00 p.m. New England Society of Physical Medicine. Hotel Kenmore, Boston.

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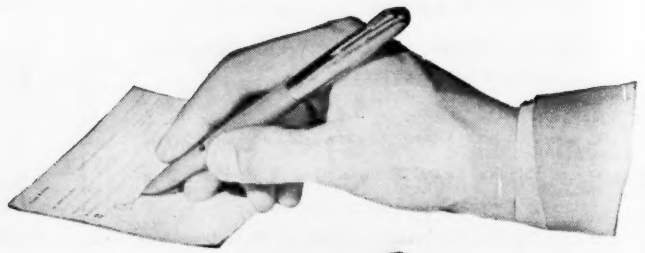

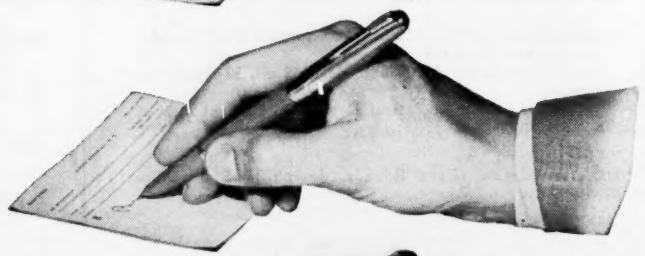

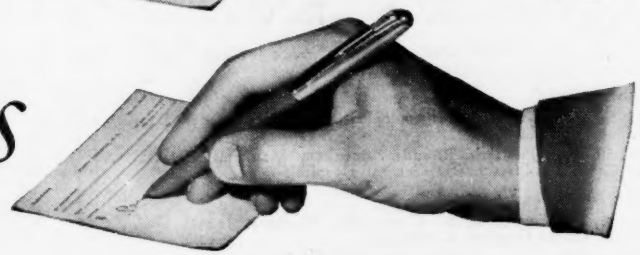

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